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RESPIRATORY CARE

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What's That Sound?

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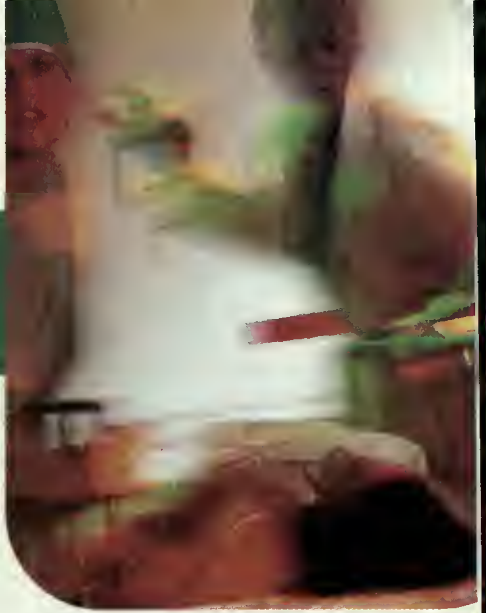

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Tobacco Control in the 21st Century: Searching for Answers in a Sea of Change—Houston T, Kaufman NJ. JAMA 2000;284:752–753.

Achieving Worldwide Tobacco Control—Brundtland GH. JAMA 2000;284:750–751.

A 36-Year-Old Woman Who Smokes Cigarettes—Rigotti NA. JAMA 2000;284:741–749.

Moving Tobacco Control Beyond "The Tipping Point": Ample Funding, Strong Policies, and "Unsticky" Cigarettes Are Key (editorial)—Davis RM. BMJ 2000;321:309-310.

Protecting Children from Passive Smoking: The Risks Are Clear and a Comprehensive Strategy Is Now Needed (editorial)—Ferrence R, Ashley MJ. BMJ 2000;321:310-311.

Improving the Treatment of Tobacco Dependence: Simple Messages and an Infrastructure to Deliver Them Are Needed (editorial)—Thun M, Glynn TJ. BMJ 2000;321:311-312.

Do Candy Cigarettes Encourage Young People to Smoke?—Klein JD, Clair SS. BMJ 2000; 321:362-365.

The Economics of Global Tobacco Control—Jha P, Chaloupka FJ. BMJ 2000;321:358-361.

Effectiveness of Interventions to Help People Stop Smoking: Findings from the Cochrane Library—Lancaster T, Stead L, Silagy C, Sowden A. BMJ 2000;321:355-358.

A Day in the Life of an Advertising Man: Review of Internal Documents from the UK Tobacco Industry's Principal Advertising Agencies—Hastings G, MacFadyen L. BMJ 2000; 321:366-371.

JAMA Patient Page: Preventing Tobacco Use in Children and Adolescents—JAMA 2000; 284:794.

US College Students' Use of Tobacco Products: Results of a National Survey—Rigotti NA, Lee JE, Wechsler H. JAMA 2000;284:699–705.

CONTEXT: Adults aged 18 to 24 years, many of whom are in college, represent the youngest legal targets for tobacco industry marketing. Cigarette smoking has been described among college students, but little is known about noncigarette tobacco use by college students or cigar use by adults of any age. OBJECTIVES: To assess the prevalence of all forms of tobacco use (cigarettes, cigars, pipes, and smokeless tobacco) among US college students and to identify student- and college-level factors associated with use of each product. DESIGN: The Harvard College Alcohol Survey, a self-administered survey conducted in 1999. SETTING: One hundred nineteen nationally representative US 4-year colleges. SUBJECTS: A total of 14,138 randomly selected students (60% response rate). MAIN OUTCOME MEASURES: Self-report of current (in the past 30 days), past-year, and lifetime use of cigarettes, cigars, pipes, smokeless tobacco, and all tobacco products. RESULTS: Nearly half (45.7%) of respondents had used a tobacco product in the past year and one third (32.9%) currently used tobacco. Cigarettes accounted for most of the tobacco use (28.5% current prevalence), but cigar use was also substantial (37.1% lifetime prevalence, 23.0% past-year prevalence, and 8.5% current prevalence) and combinations of the 2 were common. Total tobacco use was higher in men than in women (37.9% vs 29.7%; $p<0.001$), despite nearly identical current cigarette smoking rates between the sexes (28.5% for women vs 28.4% for men), because of greater use of cigars (current prevalence, 15.7% vs 3.9%; $p<0.001$) and smokeless tobacco (current prevalence, 8.7% vs 0.4%; $p<0.001$) by men. Tobacco use was significantly higher among white students ($p<0.001$), users of other substances (alcohol and marijuana) ($p<0.001$), and students whose priorities were social rather than educational or athletic ($p<0.05$). Among students who had used both cigars and cigarettes, only 8.9% smoked cigars at an earlier age than they had smoked cigarettes. CONCLUSION: Our study indicates that tobacco use is common among college students and is not limited to cigarettes. College appears to be a time when many students are trying a range of tobacco products and are in danger of developing lifelong nicotine dependence. National efforts to monitor and reduce tobacco use of all types should expand to focus on college students and other young adults.

Health Risks Associated with Cigar Smoking—Baker F, Ainsworth SR, Dye JT, Crammer C, Thun MJ, Hoffmann D. et al. JAMA 2000; 284:735–740.

This article summarizes principal findings from a conference convened by the American Cancer Society in June 1998 to examine the health risks of cigar smoking. State-of-the-science reports were presented and 120 attendees (representing government and private agencies, academia, health educators, and tobacco control experts) participated in panels and summary development discussions. The following conclusions were reached by consensus: (1) rates of cigar smoking are rising among both adults and adolescents; (2) smoking cigars instead of cigarettes does not reduce the risk of nicotine addiction; (3) as the number of cigars smoked and the amount of smoke inhaled increases, the risk of death related to cigar smoking approaches that of cigarette smoking; (4) cigar smoke contains higher concentrations of toxic and carcinogenic compounds than cigarettes and is a major source of fine-particle and carbon monoxide indoor air pollution; and (5) cigar smoking is known to cause cancers of the lung and upper aerodigestive tract.

Association Between Household and Workplace Smoking Restrictions and Adolescent Smoking—Farkas AJ, Gilpin EA, White MM, Pierce JP. JAMA 2000;284:717–722.

CONTEXT: Recent marked increases in adolescent smoking indicate a need for new prevention approaches. Whether workplace and home smoking restrictions play a role in such prevention is unknown. OBJECTIVE: To assess the association between workplace and home smoking restrictions and adolescent smoking. DESIGN, SETTING, AND SUBJECTS: Data were analyzed from 2 large national population-based surveys, the Current Population Surveys of 1992–1993 and 1995–1996, which included 17,185 adolescents aged 15 to 17 years. MAIN OUTCOME MEASURES: Smoking status of the adolescents surveyed, compared by presence of home and workplace smoking restrictions. RESULTS: After adjusting for demographics and other smokers in the household, adolescents who lived in smoke-free households were 74% (95% confidence interval [CI], 62%–88%) as likely to be smokers as adolescents who lived in households with no smoking restrictions. Similarly, adolescents who worked in smoke-free workplaces were 68% (95% CI, 51%–90%) as likely to be smokers as adolescents who worked in a workplace with no smoking restrictions. Adolescent smokers were 1.80 (95% CI, 1.23–2.65) times more likely to be former smokers if they lived in smoke-free homes. The most marked relationship of home smoking restrictions to current adolescent smoking occurred in households where all other members were never-smokers. Current smoking prevalence among adolescents in homes without smoking restrictions approached that among adolescents in homes with a current smoker but with smoking restrictions. CONCLUSIONS: Parents with minor children should be encouraged to adopt smoke-free homes. Smoke-free workplaces can also augment smoking prevention. These findings emphasize the importance of tobacco control strategies aimed at the entire population rather than at youth alone.

Smoking vs Other Risk Factors as the Cause of Smoking-Attributable Deaths: Confounding in the Courtroom—Thun MJ, Apicella LF, Henley SJ. JAMA 2000;284:706–712.

CONTEXT: The surgeon general estimates that more than 400,000 deaths are attributable to smoking annually in the United States. The tobacco industry has criticized the surgeon general's estimates because they do not control for the lower educational and socioeconomic status of modern-day smokers. OBJECTIVE: To determine whether controlling for education, occupation, race, alcohol consumption, and various dietary factors, in addition to age and sex, substantially alters the relative and attributable risk estimates associated with tobacco smoking. DESIGN, SETTING, AND PARTICIPANTS: Nationwide American Cancer Society prospective cohort study of 974,150 US adults aged 30 years or older, enrolled in 1982 and followed up through 1988. (The same study is used for the surgeon general and Centers for Disease Control and Prevention [CDC] estimates of smoking-attributable deaths in the United States.) MAIN OUTCOME MEASURES: Death from each of the chronic diseases considered in the CDC's estimate of smoking-attributable mortality (cancers of the lung, oropharynx, larynx, esophagus, pancreas, kidney, bladder, and cervix; ischemic heart disease, arterial disease, and other heart conditions; stroke; chronic obstructive pulmonary disease; and other respiratory conditions). Estimates adjusted for multiple covariates were compared with those adjusted for age only among current and former vs never smokers. RESULTS: Adjusting for multiple covariates slightly decreased the relative and attributable risk estimates for current smoking in both men and women, but slightly increased the estimates for former smoking in women. Multivariate adjustment decreased the overall estimate of deaths attributable to smoking in the United States by approximately 1%, from 401,109 to 396,741 per year. CONCLUSIONS: Our study suggests that federal estimates of deaths caused by smoking are not substantially altered by adjustment for behavioral and demographic factors associated with smoking beyond the current adjustment for age and sex.

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Changes in Youth Cigarette Use and Intentions Following Implementation of a Tobacco Control Program: Findings from the Florida Youth Tobacco Survey, 1998–2000—Bauer UE, Johnson TM, Hopkins RS, Brooks RG. JAMA 2000;284:723–728.

CONTEXT: Many states are developing tobacco use prevention and reduction programs, and current data on tobacco use behaviors and how these change over time in response to program activities are needed for program design, implementation, and evaluation. OBJECTIVES: To assess changes in youth cigarette use and intentions following implementation of the Florida Pilot Program on Tobacco Control. DESIGN, SETTING, AND PARTICIPANTS: Self-administered survey conducted prior to program implementation (1998), and 1 and 2 years (1999, 2000) later among a sample of Florida public middle school and high school students who were classified as never users, experimenters, current users, and former users of cigarettes based on survey responses. MAIN OUTCOME MEASURES: Changes in cigarette use status, intentions, and behaviors among students over a 2-year period. RESULTS: Surveys were completed by 22,540, 20,978, and 23,745 students attending 255, 242, and 243 Florida public middle and high schools in 1998, 1999 and 2000, respectively. Response rates for the 3 survey years ranged from 80% to 82% and 72% to 82% for the middle school and high school surveys, respectively. After 2 years, current cigarette use dropped from 18.5% to 11.1% ($p<0.001$) among middle school students and from 27.4% to 22.6% ($p = 0.01$) among high school students. Prevalence of never use increased from 56.4% to 69.3% ($p<0.001$) and from 31.9% to 43.1% ($p = 0.001$) among middle school and high school students, respectively. Prevalence of experimenting decreased among middle school and high school students from 21.4% to 16.2% ($p<0.001$) and from 32.8% to 28.2% ($p<0.001$), respectively. Among never users, the percentage of committed nonsmokers increased from 67.4% to 76.9% ($p<0.001$) and from 73.7% to 79.3% ($p<0.001$) among middle school and high school students, respectively. Among experimenters, the percentage of students who said they will not smoke again increased from 30.4% to 42.0% ($p<0.001$) in middle school and from 44.4% to 51.0% ($p<0.001$) in high school. CONCLUSIONS: Progress toward reduction of youth tobacco use was observed in each of the 2 years of Florida's Pilot Program on Tobacco Control. Our results suggest that a comprehensive statewide program can be effective in preventing and reducing youth tobacco use.

Smoking, Smoking Cessation, and Lung Cancer in the UK Since 1950: Combination of National Statistics with Two Case-Control Studies—Peto R, Darby S, Deo H, Silcocks P, Whitley E, Doll R. BMJ 2000;321:323-329.

Objective and design: To relate UK national trends since 1950 in smoking, in smoking cessation, and in lung cancer to the contrasting results from two large case-control studies centred around 1950 and 1990. Setting: United Kingdom. Participants: Hospital patients under 75 years of age with and without lung cancer in 1950 and 1990, plus, in 1990, a matched sample of the local population: 1465 case-control pairs in the 1950 study, and 982 cases plus 3185 controls in the 1990 study. Main outcome measures: Smoking prevalence and lung cancer. Results: For men in early middle age in the United Kingdom the prevalence of smoking halved between 1950 and 1990 but the death rate from lung cancer at ages 35-54 fell even more rapidly, indicating some reduction in the risk among continuing smokers. In contrast, women and older men who were still current smokers in 1990 were more likely than those in 1950 to have been persistent cigarette smokers throughout adult life and so had higher lung cancer rates than current smokers in 1950. The cumulative risk of death from lung cancer by age 75 (in the absence of other causes of death) rose from 6% at 1950 rates to 16% at 1990 rates in male cigarette smokers, and from 1% to 10% in female cigarette smokers. Among both men and women in 1990, however, the former smokers had only a fraction of the lung cancer rate of continuing smokers, and this fraction fell steeply with time since stopping. By 1990 cessation had almost halved the number of lung cancers that would have been expected if the former smokers had continued. For men who stopped at ages 60, 50, 40, and 30 the cumulative risks of lung cancer by age 75 were 10%, 6%, 3%, and 2%. Conclusions: People who stop smoking, even well into middle age, avoid most of their subsequent risk of lung cancer, and stopping before middle age avoids more than 90% of the risk attributable to tobacco. Mortality in the near future and throughout the first half of the 21st century could be substantially reduced by current smokers giving up the habit. In contrast, the extent to which young people henceforth become persistent smokers will affect mortality rates chiefly in the middle or second half of the 21st century.

Smoking Reduction with Oral Nicotine Inhalers: Double Blind, Randomised Clinical Trial of Efficacy and Safety—Bolliger CT, Zellweger JP, Danielsson T, van Biljon X, Robidou A, Westin A, et al. BMJ 2000;321:329-333.

Objectives: To determine whether use of an oral nicotine inhaler can result in long term reduction in smoking and whether concomitant use of nicotine replacement and smoking is safe. Design: Double blind, randomised, placebo controlled trial. Four month trial with a two year follow up. Setting: Two university hospital pulmonary clinics in Switzerland. Participants: 400 healthy volunteers, recruited through newspaper advertisements, willing to reduce their smoking but unable or unwilling to stop smoking immediately. Intervention: Active or placebo inhaler as needed for up to 18 months, with participants encouraged to limit their smoking as much as possible. Main outcome measures: Number of cigarettes smoked per day from week six to end point. Decrease verified by a measurement of exhaled carbon monoxide at each time point compared with measurement at baseline. Results: At four months sustained reduction of smoking was achieved in 52 (26%) participants in the active group and 18 (9%) in the placebo group ($p<0.001$; Fisher's test). Corresponding figures after two years were 19 (9.5%) and 6 (3.0%) ($p=0.012$). Conclusion: Nicotine inhalers effectively and safely achieved sustained reduction in smoking over 24 months. Reduction with or without nicotine substitution may be a feasible first step towards smoking cessation in people not able or not willing to stop abruptly.

Effect of Restrictions on Smoking at Home, at School, and in Public Places on Teenage smoking: Cross Sectional Study—Wakefield MA, Chaloupka FJ, Kaufman NJ, Orleans CT, Barker DC, Ruel EE. BMJ 2000;321:333-337.

Objective: To determine the relation between extent of restrictions on smoking at home, at school, and in public places and smoking uptake and smoking prevalence among school students. Design: Cross sectional survey with merged records of extent of restrictions on smoking in public places. Setting: United States. Participants: 17 287 high school students. Main outcome measures: Five point scale of smoking uptake; 30 day smoking prevalence. Results: More restrictive arrangements on smoking at home were associated with a greater likelihood of being in an earlier stage of smoking uptake ($p<0.05$) and a lower 30 day prevalence (odds ratio 0.79 (95% confidence interval 0.67 to 0.91), $p<0.001$). These findings applied even when parents were smokers. More pervasive restrictions on smoking in public places were associated with a higher probability of being in a earlier stage of smoking uptake ($p<0.05$) and lower 30 day prevalence (0.91 (0.83 to 0.99), $p=0.03$). School smoking bans were related to a greater likelihood of being in an earlier stage of smoking uptake (0.89 (0.85 to 0.99), $p<0.05$) and lower prevalence (0. 86 (0.77 to 0.94), $p<0.001$) only when the ban was strongly enforced, as measured by instances when teenagers perceived that most or all students obeyed the rule. Conclusions: These findings suggest that restrictions on smoking at home, more extensive bans on smoking in public places, and enforced bans on smoking at school may reduce teenage smoking.

Impact of the Massachusetts Tobacco Control Programme: Population Based Trend Analysis—Biener L, Harris JE, Hamilton W. BMJ 2000;321:351-354.

Objective: To assess the impact of the Massachusetts tobacco control programme, which, since its start in January 1993, has spent over \$200m-"the highest per capita expenditure for tobacco control in the world"-funded by an extra tax of 25 cents per pack of cigarettes. Design: Population based trend analysis with comparison group. Subjects: Adult residents of Massachusetts and other US states excluding California. Main outcome measures: Per capita consumption of cigarettes as measured by states' sales tax records; prevalence of smoking in adults as

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measured by several population-based telephone surveys. Results: From 1988 to 1992, decline in per capita consumption of cigarettes in Massachusetts (15%) was similar to that in the comparison states (14%), corresponding to an annual decline of 3-4% for both groups. During 1992-3, consumption continued to decline by 4% in the comparison states but dropped 12% in Massachusetts in response to the tax increase. From 1993 onward, consumption in Massachusetts showed a consistent annual decline of more than 4%, whereas in the comparison states it levelled off, decreasing by less than 1% a year. From 1992, the prevalence of adult smoking in Massachusetts has declined annually by 0.43% (95% confidence interval 0.21% to 0.66%) compared with an increase of 0. 03% (-0.06% to 0.12%) in the comparison states ($p<0.001$). Conclusions: These findings show that a strongly implemented, comprehensive tobacco control programme can significantly reduce tobacco use.

Effect of Counselling Mothers on Their Children's Exposure to Environmental Tobacco Smoke: Randomised Controlled Trial—Hovell MF, Zakarian JM, Matt GE, Hofstetter CR, Bernert JT, Pirkle J. BMJ 2000;321:337-342.

Objective: To test the efficacy of behavioural counselling for smoking mothers in reducing young children's exposure to environmental tobacco smoke. Design: Randomised double blind controlled trial. Setting: Low income homes in San Diego county, California. Participants: 108 ethnically diverse mothers who exposed their children (aged < 4 years) to tobacco smoke in the home. Intervention: Mothers were given seven counselling sessions over three months. Main outcome measures: Children's reported exposure to environmental tobacco smoke from mothers in the home and from all sources; children's cotinine concentrations in urine. Results: Mothers' reports of children's exposure to their smoke in the home declined in the counselled group from 27.30 cigarettes/week at baseline, to 4.47 at three months, to 3.66 at 12 months and in the controls from 24.56, to 12.08, to 8.38. The differences between the groups by time were significant ($p=0.002$). Reported exposure to smoke from all sources showed similar declines, with significant differences between groups by time ($p=0.008$). At 12 months, the reported exposure in the counselled group was 41.2% that of controls for mothers' smoke (95% confidence interval 34.2% to 48.3%) and was 45.7% (38.4% to 53.0%) that of controls for all sources of smoke. Children's mean urine cotinine concentrations decreased slightly in the counselled group from 10.93 ng/mL at baseline to 10.47 ng/mL at 12 months but increased in the controls from 9.43 ng/mL to 17.47 ng/mL (differences between groups by time $p=0.008$). At 12 months the cotinine concentration in the counselled group was 55.6% (48.2% to 63.0%) that of controls. Conclusions: Counselling was effective in reducing children's exposure to environmental tobacco smoke. Similar counselling in medical and social services might protect millions of children from environmental tobacco smoke in their homes.

Effects of Inverse Ratio Ventilation and Positive End-Expiratory Pressure in Oleic Acid-Induced Lung Injury—Neumann P, Berglund JE, Andersson LG, Maripu E, Magnusson A, Hedenstierna G. Am J Respir Crit Care Med 2000 May;161(5):1537-1545.

Continuous as well as cyclic (with each expiration) lung collapse in acute respiratory failure can be reduced by positive end-expiratory pressure (PEEP) or short expiration times, as in inverse ratio ventilation (IRV). In 20 pigs with oleic acid-induced lung edema, we compared the effects of a PEEP of 20 cm H_2O with IRV, using an inspiratory-to-expiratory ratio of 3:1 without external PEEP. During IRV, expiration times of 0.5 or 1.0 s were obtained with respiratory rates of 30 breaths/min or 15 breaths/min, respectively. In 15 animals, ventilation-perfusion relationships were studied through the multiple inert gas elimination technique, and lung morphology was studied with computed tomography. In another five pigs, blood flow distribution was studied with perfusion scintigraphy. All three ventilatory modes had similar effects on mean arterial blood pressure, cardiac output, oxygen delivery, and mean airway pressure. PEEP reduced shunt and improved oxygenation to a greater extent than the two modes of IRV, although there was a large variation within each group. The improvement, irrespective of which ventilatory mode was superior in a particular pig, was caused by greater and more even aeration of the lung, whereas the perfusion distribution with PEEP was the same as with IRV. Thus, the strategy of stabilizing the lungs through short expiration times, as in IRV, did not offer any advantages in our lung injury model.

Optoelectronic Plethysmography in Intensive Care Patients—Aliverti A, Dellaca R, Pelosi P, Chiumello D, Pedotti A, Gattinoni L. Am J Respir Crit Care Med 2000 May;161(5):1546-1552.

We used optoelectronic plethysmography to study 11 normal subjects during quiet and deep breathing, six sedated and paralyzed patients with acute lung injury and acute respiratory distress syndrome (ALI/ARDS) receiving continuous positive pressure ventilation (CPPV) (positive end-expiratory pressure [PEEP] = 10 cm H_2O, tidal volume [V_T] = 300, 600, 900 mL), and seven ALI/ARDS patients receiving pressure support ventilation (PSV) (PEEP 10 cm H_2O, pressure support = 5, 10, 15, 25 cm H_2O). The volumes measured using optoelectronic plethysmography were compared with measurements taken using spirometry and pneumotachography. The three methods were highly correlated. The discrepancies found were 1.7 ± 5.9%, -1.6 ± 5.4%, and 4.9 ± 6.4% when comparing optoelectronic plethysmography with spirometry, optoelectronic plethysmography with pneumotachography, and spirometry with pneumotachography, respectively. Accuracy of the compartmentalization procedure (upper thorax, lower thorax, and abdomen) was assessed by calculating compartmental volume changes during isovolume maneuvers. The discrepancy from the ideal zero line was -2.1 ± 48.3 mL. Abdominal contribution to inspired volume was greater for normal subjects than for PSV patients (63 ± 11% versus 43 ± 14%, $p < 0.001$). It decreased with V_T for normal subjects (48.5 ± 15%, $p < 0.05$), whereas it increased for CPPV patients (61 ± 10%, $p < 0.05$). No significant distribution differences were found between 5 and 25 cm H_2O PSV. We conclude that optoelectronic plethysmography is a feasible technique able to provide unique data on the distribution of chest wall volume changes in intensive care patients.

Expiratory Flow Limitation and Intrinsic Positive End-Expiratory Pressure at Zero Positive End-Expiratory Pressure in Patients with Adult Respiratory Distress Syndrome—Koutsoukou A, Armaganidis A, Stavrakaki-Kallergi C, Vassilakopoulos T, Lymberis A, Roussos C, Milic-Emili J. Am J Respir Crit Care Med 2000 May;161(5):1590-1596.

It has been suggested that in patients with adult respiratory distress syndrome (ARDS), intrinsic positive end-expiratory pressure (PEEPi) is generated by a disproportionate increase in expiratory flow resistance. Using the negative expiratory pressure (NEP) technique, we assessed whether expiratory flow limitation (EFL) and PEEPi were present at zero PEEP in 10 semirecumbent, mechanically ventilated ARDS patients. Because bronchodilators may decrease airway resistance, we also investigated the effect of nebulized salbutamol on EFL, PEEPi, and respiratory mechanics in these patients, and in seven patients we measured the latter variables in the supine position as well. In the semirecumbent position, eight of the 10 ARDS patients exhibited tidal EFL, ranging from 5 to 37% of the control tidal volume (V_T), whereas PEEPi was present in all 10 subjects, ranging from 0.4 cm H_2O to 7.7 cm H_2O. The onset of EFL was heralded by a distinct inflection point on the expiratory flow-volume curve, which probably reflected small-airway closure. Administration of salbutamol had no statistically significant effect on PEEPi, EFL (as $\%V_2$), or respiratory mechanics. EFL ($\%V_2$) and PEEPi were significantly higher in the supine position than in the semirecumbent position, whereas the other respiratory variables did not change. Our results suggest that in the absence of externally applied PEEP, most ARDS patients exhibit EFL associated with small-airway closure and a concomitant PEEPi.

Predictors of Mortality in Acute Respiratory Distress Syndrome: Focus on the Role of Right Heart Catheterization—Vieillard-Baron A, Girou E, Valente E, Brun-Buisson C, Jardin F, Lemaire F, Brochard L. Am J Respir Crit Care Med 2000 May;161(5):1597-1601.

Right heart catheterization (RHC) has been suspected of increasing mortality. The acute respiratory distress syndrome (ARDS) is a frequent reason for RHC. We designed a retrospective cohort study of 119 consecutive ARDS patients admitted to two medical intensive care units of tertiary care hospitals in which two different approaches are used for hemodynamic monitoring: RHC on demand (Henri Mondor Hospital [HM]) and no use of RHC (Ambroise Pare Hospital [AP]). The study tried to identify risk factors for death, and to assess the influence of RHC, with adjustment for the intensity of hemodynamic support as a confounding factor, using 98 patients in whom the delay between onset of ARDS, use of vasopressors, and RHC did not exceed 48 h. Several variables, including septic shock, cause of ARDS, Simplified Acute Physiology Score (SAPS) II, use of epinephrine/norepinephrine, and presence of RHC were entered into a logistic regression model to evaluate their independent prognostic roles. Mortality was different at HM and AP (36 of 55 patients [65.5%] versus 16 of 43 patients [37.2%], $p < 0.005$), and 29 of the 35 RHC-monitored patients died (82.8%), as compared with 23 of 63 patients (36.5%) treated without RHC ($p < 0.0001$). However, administration of epinephrine/norepinephrine and a nonpulmonary cause of ARDS were each independently associated with death. It is only when administration of vasopressors was omitted from the model that RHC, septic shock, and SAPS II became independent predictors of mortality. These results suggest that: (1) the use of vasopressors, but not of RHC, represents an important prognostic factor; and (2) not taking into account the use of these drugs may be misleading when assessing the influence of RHC on outcome.

Overcoming the Adverse Effect of Humidity in Aerosol Delivery via Pressurized Metered-Dose Inhalers during Mechanical Ventilation—Lange CF, Finlay WH. Am J Respir Crit Care Med 2000 May;161(5): 1614-1618.

The well-known problem of reduced drug delivery that occurs when heated, humid air is used with pressurized metered-dose inhalers (pMDIs) and spacers in intubated settings is carefully studied with Airomir using an in vitro model under controlled conditions of temperature and humidity. A better understanding of the physical processes leading to the aforementioned drop in performance is obtained, and a method is devised to circumvent the problem without having to reduce the temperature or humidity of the ventilator circuit. The present study shows that the mole fraction of water vapor in the ventilation air (and not the temperature) is the major factor behind the sharp drop in the amount of drug delivered to the lung. However, the presence of water vapor does not affect performance because of hygroscopic growth. Instead, it influences the initial atomization process and the early stages of aerosol generation. Removal of these negative effects can be achieved by using a larger spacer that allows longer times for the aerosol to evaporate, as is demonstrated in the present study.

The Prone Position Eliminates Compression of the Lungs by the Heart—Albert RK, Hubmayr RD. Am J Respir Crit Care Med 2000 May;161(5):1660-1665.

The prone position improves gas exchange in many patients with ARDS. Animal studies have indicated that turning prone restores ventilation to dorsal lung regions without markedly compromising ventral regions. To investigate a potential mechanism by which this might occur, the relative volume of lung located directly under the heart was measured in the supine and prone positions in seven patients. Four axial tomographic sections between the carina and the diaphragm were analyzed (Sections 1 through 4). When supine, the percent of the total lung volume located under the heart increased from 7 ± 4% to 42 ± 8%, and from 11 ± 4% to 16 ± 4% in Sections 1 through 4, in the left and right lungs, respectively. When prone, the percent of left and right lung volume located under the heart was ≤ 1 and ≤ 4 %, respectively, in all four sections ($p < 0.05$ for each section, supine versus prone). Although a large fraction of the lung, particularly on the left, is located directly under the heart in supine patients, and would be subject to the compressive force resulting from heart weight, almost no lung is located under the heart when patients are prone and the compressive force of the heart is directed toward the sternum.

Airway Hyperresponsiveness in Elite Athletes—Langdeau JB, Turcotte H, Bowie DM, Jobin J, Desgagne P, Boulet LP. Am J Respir Crit Care Med 2000 May;161(5):1479-1484.

It has been suggested that high-level training could contribute to the development of airway hyperresponsiveness (AHR), but the comparative effects of different sports on airway function remains to be determined. We evaluated 150 nonsmoking volunteers 18 to 55 yr of age; 100 athletes divided into four subgroups of 25 subjects each according to the predominant estimated hydrocaloric characteristic of ambient air inhaled during training: dry air (DA), cold air (CA), humid air (HA) and a mixture of dry and humid air (MA), and 50 sedentary subjects. Each subject had a respiratory questionnaire, a methacholine challenge, allergy skin-prick tests, and heart rate variability recording for evaluation of parasympathetic tone. The athletes had a 49% prevalence of AHR ($PC_{20} <$ 16 mg/mL), with a mean PC_{20} of 16.9 mg/mL, compared with 28% (PC_{20}: 35.4) in sedentary subjects ($p = 0.009$). The prevalence (%) of AHR and mean PC_{20} (mg/mL) varied as followed in the four subgroups of athletes: DA: 32% and 30.9; CA: 52% and 15.8; HA: 76% and 7.3; and MA: 32% and 21.5 ($p = 0.002$). The estimated parasympathetic tone was higher in athletes ($p < 0.001$), but this parameter showed only a weak correlation with PC_{20} ($r = -0.17$, $p = 0.04$). This study has shown a significantly higher prevalence of AHR in athletes than in the control group because of the higher prevalence in the CA and HA groups. Parasympathetic activity may act as modulator of airway responsiveness, but the increased prevalence of AHR in our athlete population may be related to the type and possibly the content of inhaled air during training.

Are Elderly Patients with Obstructive Airway Disease Being Prematurely Discharged?—Sin DD, Tu JV. Am J Respir Crit Care Med 2000 May;161(5):1513-1517.

Despite the temporal trend toward decreasing length of hospital stay for all medical conditions in North America, the effect of different lengths of hospitalization on short-term outcomes such as readmission or mortality has not been well studied. However, there is growing concern that very short stays in hospital may result in premature discharges, which may lead to worse outcomes for patients. We conducted a population-based study of elderly patients with obstructive airway disease in Ontario, Canada to test the hypothesis that very short initial hospital stays increase the short-term risk for readmission and mortality. Using a cohort of 32,384 elderly patients 65 yr of age or older, we compared 15-d rates of readmission and mortality among patients with different lengths of stay. Although patients with hospital stays of less than 4 d were younger and had fewer comorbidities, they were 39% (95% confidence interval [[CI], 20% to 61%) more likely to be readmitted and 45% (95% CI, 9% to 92%) more likely to die within 15 d postdischarge compared with those who stayed 4 to 6 d. The risk was highest among patients whose stay was less than or equal to 1 hospital day; they had a 69% (95% CI, 32% to 117%)

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excess risk of readmission and a 2.08 (95% CI, 1.23 to 3.45) -fold increase in mortality compared with those who stayed in hospital for 2 d. This suggests that some elderly patients with obstructive airway disease may be being prematurely discharged.

The Role of Hypoventilation and Ventilation-Perfusion Redistribution in Oxygen-Induced Hypercapnia during Acute Exacerbations of Chronic Obstructive Pulmonary Disease—Robinson TD, Freiberg DB, Regnis JA, Young IH. Am J Respir Crit Care Med 2000 May;161(5): 1524-1529.

The detailed mechanisms of oxygen-induced hypercapnia were examined in 22 patients during an acute exacerbation of chronic obstructive pulmonary disease. Ventilation, cardiac output, and the distribution of ventilation-perfusion ($\dot{V}_A/\dot{Q}$) ratios were measured using the multiple inert gas elimination technique breathing air and then 100% oxygen through a nose mask. Twelve patients were classified as retainers (R) when P_{aCO_2} rose by more than 3 mm Hg (8.3 ± 5.6; mean ± SD) after breathing 100% oxygen for at least 20 min. The other 10 patients showed a change in P_{aCO_2} of -1.3 ± 2.2 mm Hg breathing oxygen and were classified as nonretainers (NR). Ventilation fell significantly from 9.0 ± 1.5 to 7.2 ± 1.2 L/min in the R group breathing oxygen ($p = 0.007$), whereas there was no change in ventilation in the NR group (9.8 ± 1.8 to 9.9 ± 1.8 L/min). The dispersion of $\dot{V}_A/\dot{Q}$ ratios as measured by log SD of blood flow (log SD $\dot{Q}$) increased significantly in both R (0.96 ± 0. 17 to 1.13 ± 0.17) and NR (0.77 ± 0.20 to 1.04 ± 0.23, $p < 0.05$) groups breathing oxygen, whereas log SD of ventilation (log SD $\dot{Q}$) increased only in the R group (0.97 ± 0.24 to 1.20 ± 0.46, $p < 0.05$). This study suggests that an overall reduction in ventilation characterizes oxygen-induced hypercapnia, as an increased dispersion of blood flow from release of hypoxic vasoconstriction occurred to a significant and similar degree in both groups. The significant increase in wasted ventilation (alveolar dead space) in the R group only may be secondary to the higher carbon dioxide tension, perhaps related to bronchodilatation.

Time Course and Recovery of Exacerbations in Patients with Chronic Obstructive Pulmonary Disease—Seemungal TA, Donaldson GC, Bhowmik A, Jeffries DJ, Wedzicha JA. Am J Respir Crit Care Med 2000 May;161(5):1608-1613.

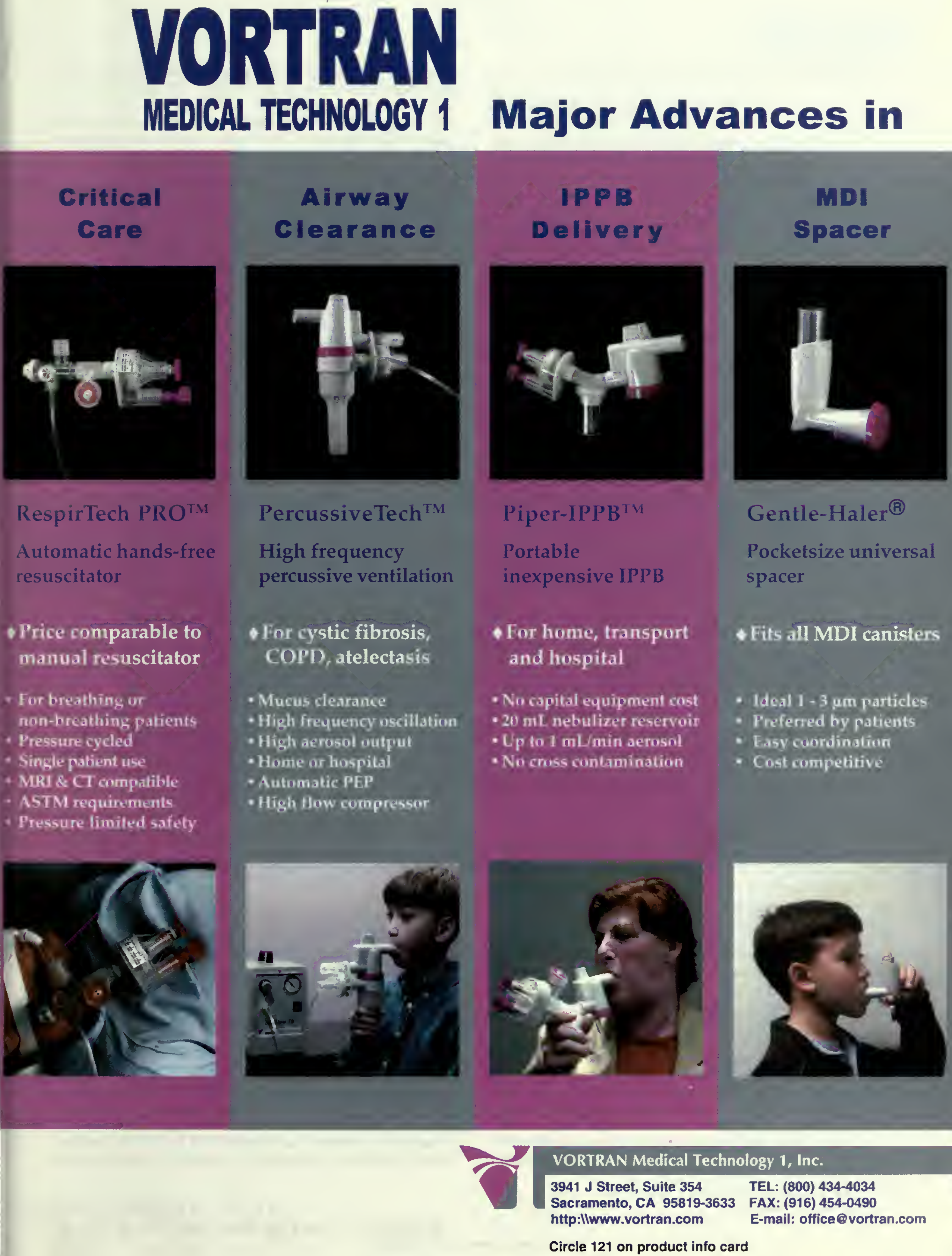
Although exacerbations of chronic obstructive pulmonary disease (COPD) are associated with symptomatic and physiological deterioration, little is known of the time course and duration of these changes. We have studied symptoms and lung function changes associated with COPD exacerbations to determine factors affecting recovery from exacerbation. A cohort of 101 patients with moderate to severe COPD (mean FEV_1 41.9% predicted) were studied over a period of 2.5 yr and regularly followed when stable and during 504 exacerbations. Patients recorded daily morning peak expiratory flow rate (PEFR) and changes in respiratory symptoms on diary cards. A subgroup of 34 patients also recorded daily spirometry. Exacerbations were defined by major symptoms (increased dyspnea, increased sputum purulence, increased sputum volume) and minor symptoms. Before onset of exacerbation there was deterioration in the symptoms of dyspnea, sore throat, cough, and symptoms of a common cold (all $p < 0.05$), but not lung function. Larger falls in PEFR were associated with symptoms of increased dyspnea ($p = 0.014$), colds ($p = 0.047$), or increased wheeze ($p = 0.009$) at exacerbation. Median recovery times were 6 (interquartile range [IQR] 1 to 14) d for PEFR and 7 (IQR 4 to 14) d for daily total symptom score. Recovery of PEFR to baseline values was complete in only 75.2% of exacerbations at 35 d, whereas in 7.1% of exacerbations at 91 d PEFR recovery had not occurred. In the 404 exacerbations where recovery of PEFR to baseline values was complete at 91 d, increased dyspnea and colds at onset of exacerbation were associated with prolonged recovery times ($p < 0.001$ in both cases). Symptom changes during exacerbation do not closely reflect those of lung function, but their increase may predict exacerbation, with dyspnea or colds characterizing the more severe. Recovery is incomplete in a significant proportion of COPD exacerbations.

Effects of Hospitalists on Cost, Outcomes, and Patient Satisfaction in a Rural Health System—Davis KM, Koch KE, Harvey JK, Wilson R, Englert J, Gerard PD. Am J Med 2000 Jun 1;108(8):621-626.

PURPOSE: Previous studies have examined the effects of hospitalists in urban academic hospitals. We compared the outcomes of patients treated by hospitalists with those of patients treated by internists at a 647-bed rural community hospital. SUBJECTS AND METHODS: The 443 patients in the hospitalists' 10 most common diagnosis-related groups (DRGs) were compared with 1,681 patients in the same DRGs who were cared for by internists in fiscal year 1998. Length of stay, cost of care, patient illness severity, patient satisfaction, 30-day readmission rate, inpatient mortality, discharge status, and resource utilization were compared.RESULTS: The hospitalists' patients had a shorter mean (± SD) length of stay (4.1 ± 3.0 days versus 5.5 ± 4.9 days, $p < 0.001$) and their cost of care was less than that of the internists' patients ($4,098 ± $2,455 versus $4,658 ± $4,084, $p < 0.001$). Analyses that adjusted for patient age, race, sex, insurance status, severity of illness, and specific medical comorbidities confirmed these differences. The differences between hospitalists and internists were most apparent among very ill patients. Mortality rates were similar (4.5% for hospitalists versus 4.9% for internists, $p = 0.80$), as were the readmission rates (4.5% for hospitalists versus 5.6% for internists, $p = 0.41$). Patient satisfaction was similar for both groups. The internists used more resources in 8 of 11 categories. CONCLUSIONS: The hospitalists provided cost-effective care, particularly for the sickest patients, with good outcomes and patient satisfaction.

Early Use of Non-Invasive Ventilation for Acute Exacerbations of Chronic Obstructive Pulmonary Disease on General Respiratory Wards: A Multicentre Randomised Controlled Trial—Plant PK, Owen JL, Elliott MW. Lancet 2000 Jun 3;355(9219):1931-1935.

BACKGROUND: Within the intensive-care unit, non-invasive ventilation (NIV) can prevent the need for intubation and the mortality associated with severe episodes of chronic obstructive pulmonary disease (COPD). The aim of this study was to find whether the introduction of NIV, early after the admission on a general respiratory ward, was effective at reducing the need for intubation and the mortality associated with acute exacerbations of COPD. METHODS: We did a prospective multicentre randomised controlled study comparing NIV with standard therapy in patients with mild to moderate acidosis. NIV was administered on the ward with a simple non-invasive ventilator and a standardised predefined protocol. Patients were recruited from 14 UK hospitals over 22 months. FINDINGS: 236 patients were recruited, 118 received standard therapy alone and 118 additional NIV. The two groups had similar characteristics at enrolment. The use of NIV significantly reduced the need for intubation as defined by the failure criteria. 32/118 (27%) of the standard group failed compared with 18/118 (15%) of the NIV group ($p=0.02$). In-hospital mortality was also reduced by NIV, 24/118 (20%) died in the standard group compared with 12/118 (10%) in the NIV group ($p=0.05$). In both groups pH, P_{aCO_2}, and respiratory rate improved at 4 h ($p<0.01$). However, NIV led to a more rapid improvement in pH in the first hour ($p=0.02$) and a greater fall in respiratory rate at 4 h ($p=0.035$). The duration of breathlessness was also reduced by NIV ($p=0.025$). INTERPRETATION: The early use of NIV for mildly and moderately acidotic patients with COPD in the general ward setting leads to more rapid improvement of physiological variables, a reduction in the need for invasive mechanical ventilation (with objective criteria), and a reduction in in-hospital mortality.



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Use of Inhaled Nitric Oxide. Pediatrics 2000;106:344-345.

Approval of inhaled nitric oxide by the US Food and Drug Administration for hypoxic respiratory failure of the term and near-term newborn provides an important new therapy for this serious condition. This statement addresses the conditions under which inhaled nitric oxide should be administered to the neonate with hypoxic respiratory failure.

High-Dose Albuterol by Metered-Dose Inhaler Plus a Spacer Device Versus Nebulization in Preschool Children with Recurrent Wheezing: A Double-Blind, Randomized Equivalence Trial—Ploin D, Chapuis FR, Stamm D, Robert J, David L, Chatelain PG, et al. Pediatrics 2000;106:311-317.

Inhaled albuterol is the most frequently used bronchodilator for acute wheezing, and nebulization is the standard mode of delivery in hospital setting. However, recent guidelines consider spacer devices as an easier to use, and cost-saving alternative and recommend the high-dose metered-dose inhaler bronchodilator. Objective. To demonstrate clinical equivalence between a spacer device and a nebulizer for albuterol administration. Design. Randomized, double-blind, parallel group equivalence trial. Setting. Pediatric emergency wards at 2 tertiary teaching hospitals. Patients. Sixty-four 12- to 60-month-old children with acute recurrent wheezing (32 per group). Interventions. Albuterol was administered through the spacer device (50 mcg/kg) or through the nebulizer (150 mcg/kg) and repeated 3 times at 20-minute intervals. Parents completed a questionnaire. Outcome Measures. Pulmonary index, hospitalization, ease of use, acceptability, and pulse oximetry saturation. Results. The 90% confidence interval of the difference between treatment groups for the median absolute changes in pulmonary index values between T0 and T60 was [-1; +1] and was included in the equivalence interval [-1.5; +1.5]. Clinical improvement increased with time. Less than 10% of the children (3 in each group) required hospitalization (2 in each group attributable to treatment failure). Parents considered administration of albuterol using the spacer device easier (94%) and better accepted by their children (62%). Conclusions. The efficacy of albuterol administered using the spacer device was equivalent to that of the nebulizer. Given its high tolerance, repeated 50-mcg/kg doses of albuterol administered through the spacer device should be considered in hospital emergency departments as first-line therapy for wheezing.

Effect of an Additional 1-Hour T-Piece Trial on Weaning Outcome at Minimal Pressure Support—Koh Y, Hong SB, Lim CM, Lee SD, Kim WS, Kim DS, Kim WD. J Crit Care 2000;15:41-45.

PURPOSE: The purpose of this article was to investigate the effect of an additional 1-hour T-piece trial at the level of minimum pressure support (PS_{min}) on weaning outcome. MATERIALS AND METHODS: Open, randomized, prospective study of 44 patients who had received mechanical ventilation for more than 3 days.Thirty-six patients satisfied the protocol. There were 42 weaning trials. The patients were randomized into an intervention group (additional 1-hour period of T-piece trial) and a control group (extubation directly) at PS_{min}. Blood gas analysis and estimation of respiratory and hemodynamic variables were performed at the 15 cm H_2O level of pressure support. Measurements were repeated at PS_{min} and during weaning process (in intervention group). RESULTS: Mean PS_{min} level was 7.6 (± 1.9) cm H_2O. There were no differences in total ventilation time (TVT), acute physiology and chronic health evaluation (APACHE) II score, nutritional indices, and respiratory mechanics on PS_{min} between the two groups. The weaning success rate and the reintubation rate were similar for the intervention group (55% and 18%, respectively) and control group (70% and 20%, respectively). Work of breathing, pressure time product, and tidal volume significantly worsened after a 1-hour T-piece trial when compared with those values measured at PS_{min} in the intervention group ($p < 0.05$). For the combined patient sample, TVT and tidal volume at PS_{min} were significantly different between the patients with weaning success (246 ± 195 hours, 0.43 ± 0.11 L) and those with weaning failure (407 ± 248 hours, 0.35 ± 0.10 L) ($p < 0.05$ in each). CONCLUSION: There were no advantages in weaning outcome by the addition of a 1-hour T-piece trial compared with prompt extubation at PS_{min}.

The Impact of Respiratory Variables on Mortality in Non-ARDS and ARDS Patients Requiring Mechanical Ventilation—Luhr OR, Karlsson M, Thorsteinsson A, Rylander C, Frostell CG. Intensive Care Med 2000;26:508-517.

OBJECTIVES: Primarily, to determine if respiratory variables, assessed on a daily basis on days 1-6 after ICU admission, were associated with mortality in non-ARDS and ARDS patients with respiratory failure requiring mechanical ventilation. Secondarily, to determine non-respiratory factors associated with mortality in ARDS and non-ARDS patients. DESIGN: Prospective multicentre clinical study. SETTING: Seventy-eight intensive care units in Sweden and Iceland. PATIENTS: Five hundred twenty non-ARDS and 95 ARDS patients. MEASUREMENTS AND RESULTS: Potentially prognostic factors present at inclusion were tested against 90-day mortality using a Cox regression model. Respiratory variables (P_{aO_2}/F_{IO_2}, PEEP, mean airway pressure (MAP) and base excess (BE)) were tested against mortality using the model. Primary aim: in non-ARDS a low P_{aO_2}/F_{IO_2} on day 1, RR (risk ratio) = 1.17, CI (95% confidence interval) (1.00; 1.36), day 4, 1.24 (1.02; 1.50), day 5, 1.25 (1.02; 1.53) and a low MAP at baseline, 1.18 (1.00; 1.39), day 2, 1.24 (1.02; 1.52), day 3, 1.33 (1.06; 1.67), day 6, 2.38 (1.11; 5.73) were significantly associated with 90-day death. Secondary aim: in non-ARDS a low age, RR = 0.77 (0.67; 0.89), female gender, 0.85 (0.74; 0.98), and low APS (acute physiologic score), 0.85 (0.73; 0.99), were associated with survival; chronic disease, 1.31 (1.12; 1.52), and non-pulmonary origin to the respiratory failure, 1.27 (1.10; 1.47), with death. In ARDS low age, RR = 0.65 CI (0.46; 0.91), and low APS, 0.65 (0.46; 0.90), were associated with survival. CONCLUSIONS: No independent significant association was seen between 90-day mortality and degree of hypoxaemia, PEEP, MAP or BE for the first full week of ICU care in either ARDS or non-ARDS. In a sub-group of non-ARDS a lower P_{aO_2}/F_{IO_2} and MAP tended to influence mortality where a significant association was seen for 3 of 7 study days. Age, gender, APS, presence of a chronic disease and a pulmonary/non-pulmonary reason for the respiratory failure were associated with mortality in non-ARDS, while only age and APS showed a similar association in ARDS.

Effects of Periodic Lung Recruitment Maneuvers on Gas Exchange and Respiratory Mechanics in Mechanically Ventilated Acute Respiratory Distress Syndrome (ARDS) Patients—Foti G, Cereda M, Sparacino ME, De Marchi L, Villa F, Pesenti A. Intensive Care Med 2000; 26:501-507.

OBJECTIVE: We wished to investigate whether volume recruitment maneuvers (VRMs) could improve alveolar recruitment and oxygenation in acute respiratory distress syndrome (ARDS) patients, ventilated at relatively low positive end-expiratory pressure (PEEP). SETTING: General intensive care unit (ICU) located in a teaching hospital. PATIENTS: 15 PEEP responder ARDS patients undergoing continuous positive pressure ventilation (CPPV) with sedation and muscle paralysis. INTERVENTIONS: We identified a low (9.4 ± 3 cmH_2O) and a high (16.0 ± 2 cmH_2O) level of PEEP associated with target oxygenation values. Using a custom modified mechanical ventilator, we applied in random order three steps lasting 30 min: (1) CPPV at the low PEEP level ($CPPV_{LO}$); (2) CPPV at the high PEEP level ($CPPV_{HI}$); (3) CPPV at low PEEP with the superimposition of periodic VRMs ($CPPV_{VRM}$). VRMs were performed twice a minute by increasing PEEP to the high level for two breaths. Each brace of two breaths was spaced 30 seconds from the

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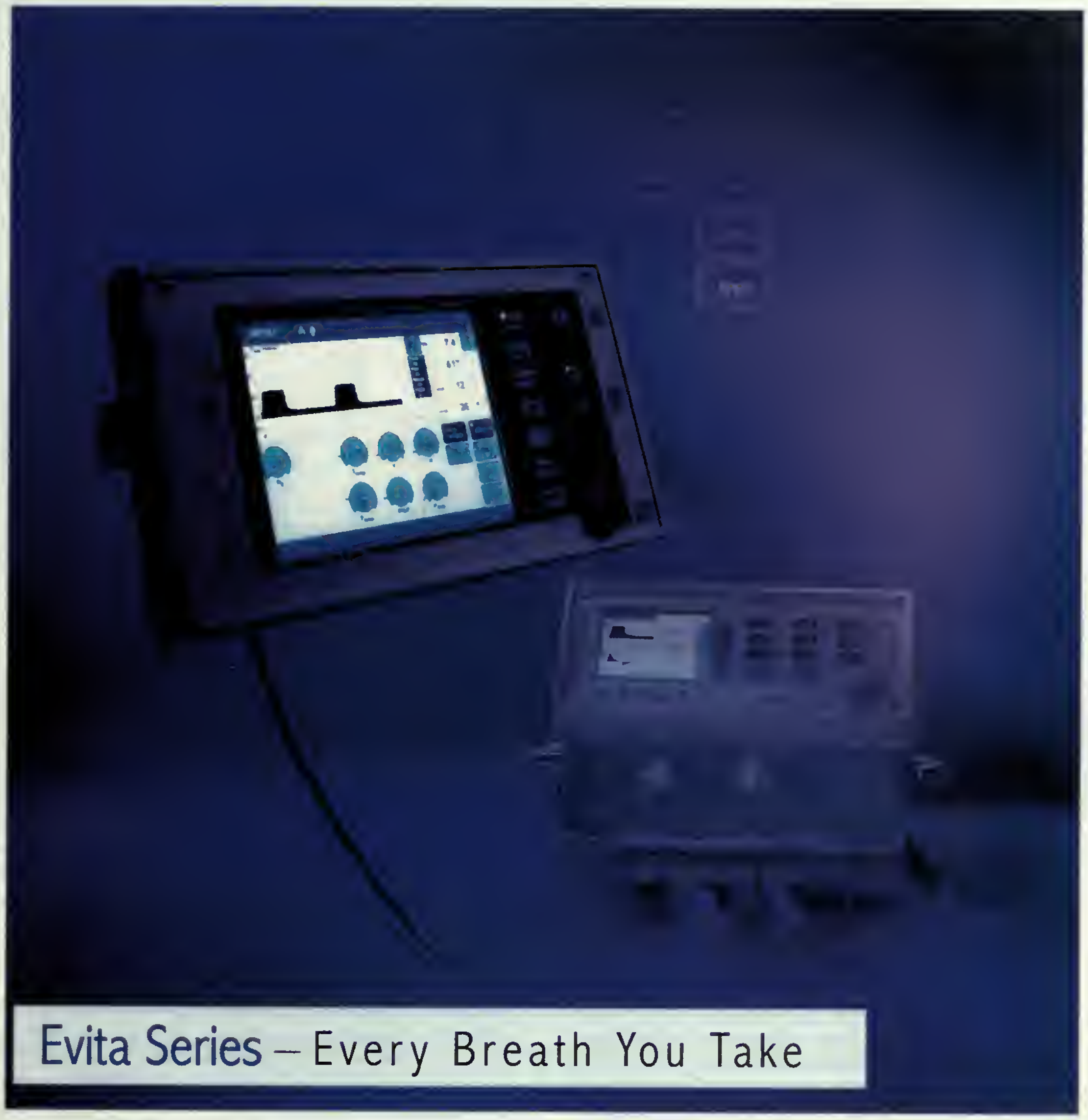

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preceding one. MEASUREMENTS AND RESULTS: We measured gas exchange, hemodynamics, respiratory mechanics, and the end expiratory lung volume (EELV). Compared to $CPPV_{LO}$, $CPPV_{VRM}$ resulted in higher P_{aO_2} (117.9 ± 40.6 vs 79.4 ± 13.6 mm Hg, $p < 0.01$) and EELV (1.50 ± 0.62 vs 1.26 ± 0.50 l, $p < 0.05$), and in lower venous admixture ($\dot{Q}_{VA}/\dot{Q}_T$) (0.42 ± 0.07 vs 0.48 ± 0.07, $p < 0.01$). During $CPPV_{HI}$, we observed significantly higher P_{aO_2} (139.3 ± 32.5 mm Hg) and lower $\dot{Q}_{VA}/\dot{Q}_T$ (0.37 ± 0.08) compared to $CPPV_{LO}$ ($p < 0.01$) and to $CPPV_{VRM}$ ($p < 0.05$). CONCLUSIONS: VRMs can improve oxygenation and alveolar recruitment during CPPV at relatively low PEEP, but are relatively less effective than a continuous high PEEP level.

The Increase in CO_2 Production Induced by $NaHCO_3$ Depends on Blood Albumin and Hemoglobin Concentrations—Levraut J, Garcia P, Giunti C, Ichai C, Bouregba M, Ciebiera JP, et al. Intensive Care Med 2000;26:558-564.

OBJECTIVE: To evaluate the origin of H^+ ions participating in the generation of CO_2 coming from sodium bicarbonate infusion during metabolic acidosis. We hypothesized that these H^+ ions come from a back-titration of the main non-bicarbonate buffers present in the blood, i. e. the hemoglobin and the albumin, and thus postulated that the rate of CO_2 release from a bicarbonate load is dependent on the concentration of these buffers. DESIGN: Prospective clinical and experimental study. SETTING: Surgical intensive care unit of a university hospital. PATIENTS AND MATERIAL: (1) Sixteen stable sedated and artificially ventilated critically ill patients with a mild base deficit. (2) Acidotic human blood (bicarbonate 5 mM, pH 7.0) of hematocrit 5, 10, 20 and 40% regenerated from a mixture of frozen fresh plasma and packed red blood cells. INTERVENTIONS: Patients: infusion of 1.5 mmol/kg sodium bicarbonate over 5 min. Regenerated blood: 25 mM sodium bicarbonate load. MEASUREMENTS AND RESULTS: Patients: continuous measurement of CO_2 production ($\dot{V}_{CO_2}$) on the expired gas using a metabolic monitor and arterial blood gas analysis before (T0), at the end (T5) and at 10, 30 and 60 min after the beginning of the bicarbonate infusion. The increase in $\dot{V}_{CO_2}$ was 18 ± 7% leading to a rise in P_{aCO_2} from 39.6 ± 2.3 at T0 to 46.2 ± 2.7 mm Hg at T5. The increases in $\dot{V}_{CO_2}$ and in P_{aCO_2} were significantly correlated to the albumin ($r = 0.73$, $p < 0.005$ and $r = 0.70$, $p < 0.005$, respectively) and to the hemoglobin ($r = 0.51$, $p < 0.05$ and $r = 0.65$, $p < 0.01$, respectively) concentrations. Regenerated blood: gas analysis 1 min after the bicarbonate load. The increase in P_{CO_2} was closely related to the hematocrit (Ht) of the blood as it was 15.9 ± 7.5 mm Hg for Ht 5%, 29.0 ± 9.6 for Ht 10%, 44.2 ± 5.9 for Ht 20% and 71.0 ± 3.5 for Ht 40% ($n = 5$ for each, $p < 0.001$). CONCLUSIONS: The importance of the release of CO_2 from a bicarbonate load is dependent on the concentration of the blood non-bicarbonate buffers. It is therefore likely that the adverse effects of bicarbonate therapy linked to the CO_2 generation are more important in patients with high blood albumin and hemoglobin concentrations.

Policy Statement: Recommendations for the Prevention of Pneumococcal Infections, Including the Use of Pneumococcal Conjugate Vaccine (Prevnar), Pneumococcal Polysaccharide Vaccine, and Antibiotic Prophylaxis. Pediatrics 2000;106:362-366.

Heptavalent pneumococcal conjugate vaccine (PCV7) is recommended for universal use in children 23 months and younger, to be given concurrently with other recommended childhood vaccines at 2, 4, 6, and 12 to 15 months of age. For children 7 to 23 months old who have not received previous doses of PCV7, administration of a reduced number of doses is recommended. Two doses of PCV7 are recommended for children 24 to 59 months old at high risk of invasive pneumococcal infection-including children with functional, anatomic, or congenital asplenia; infection with human immunodeficiency virus; and other predisposing conditions-who have not been immunized previously with PCV7. Recommendations have been made for use of 23-valent pneumococcal polysaccharide (23PS) vaccine in high-risk children to expand serotype coverage. High-risk children should be given vaccines at the earliest possible opportunity. Use of antibiotic prophylaxis in children younger than 5 years with functional or anatomic asplenia, including children with sickle cell disease, continues to be recommended. Children who have not experienced invasive pneumococcal infection and have received recommended pneumococcal immunizations may discontinue prophylaxis after 5 years of age. The safety and efficacy of PCV7 and 23PS in children 24 months or older at moderate or lower risk of invasive pneumococcal infection remain under investigation. Current US Food and Drug Administration indications are for administration of PCV7 only to children younger than 24 months. Data are insufficient to recommend routine administration of PCV7 for children at moderate risk of pneumococcal invasive infection, including all children 24 to 35 months old, children 36 to 59 months old who attend out-of-home care, and children 36 to 59 months old who are of Native American (American Indian and Alaska Native) or African American descent. However, all children 24 to 59 months old, regardless of whether they are at low or moderate risk, may benefit from the administration of pneumococcal immunizations. Therefore, a single dose of PCV7 or 23PS vaccine may be given to children 24 months or older. The 23PS is an acceptable alternative to PCV7, although an enhanced immune response and probable reduction of nasopharyngeal carriage favor the use of PCV7 whenever possible.

Technical Report: Prevention of Pneumococcal Infections, Including the Use of Pneumococcal Conjugate and Polysaccharide Vaccines and Antibiotic Prophylaxis—Overturf GD. Pediatrics 2000;106:367-376.

Pneumococcal infections are the most common invasive bacterial infections in children in the United States. The incidence of invasive pneumococcal infections peaks in children younger than 2 years, reaching rates of 228/100 000 in children 6 to 12 months old. Children with functional or anatomic asplenia (including sickle cell disease [SCD]) and children with human immunodeficiency virus infection have pneumococcal infection rates 20- to 100-fold higher than those of healthy children during the first 5 years of life. Others at high risk of pneumococcal infections include children with congenital immunodeficiency; chronic cardiopulmonary disease; children receiving immunosuppressive chemotherapy; children with immunosuppressive neoplastic diseases; children with chronic renal insufficiency, including nephrotic syndrome; children with diabetes; and children with cerebrospinal fluid leaks. Children of Native American (American Indian and Alaska Native) or African American descent also have higher rates of invasive pneumococcal disease. Outbreaks of pneumococcal infection have occurred with increased frequency in children attending out-of-home care. Among these children, nasopharyngeal colonization rates of 60% have been observed, along with pneumococci resistant to multiple antibiotics. The administration of antibiotics to children involved in outbreaks of pneumococcal disease has had an inconsistent effect on nasopharyngeal carriage. In contrast, continuous penicillin prophylaxis in children younger than 5 years with SCD has been successful in reducing rates of pneumococcal disease by 84%. Pneumococcal polysaccharide vaccines have been recommended since 1985 for children older than 2 years who are at high risk of invasive disease, but these vaccines were not recommended for younger children and infants because of poor antibody response before 2 years of age. In contrast, pneumococcal conjugate vaccines (Prevnar) induce proposed protective antibody responses (> 0.15 mcg/mL) in >90% of infants after 3 doses given at 2, 4, and 6 months of age. After priming doses, significant booster responses (ie, immunologic memory) are apparent when additional doses are given at 12 to 15 months of age. In efficacy trials, infant immunization with Prevnar decreased invasive infections by >93% and consolidative pneumonia by 73%, and it was associated with a 7% decrease in otitis media and a 20% decrease in tympanostomy tube placement. Adverse events after the administration of Prevnar have been

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[2] Raoof, S., et. al., *Chest*, Vol. 115. No. 6, pp. 1658-1666, June 1999

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limited to areas of local swelling or erythema of 1 to 2 cm and some increase in the incidence of postimmunization fever when it is given with other childhood vaccines. Based on data in phase 3 efficacy and safety trials, the US Food and Drug Administration has provided an indication for the use of Prevnar in children younger than 24 months.

Volume-Outcome Relationships in Pediatric Intensive Care Units—Tilford JM, Simpson PM, Green JW, Lensing S, Fiser DH. Pediatrics 2000;106:289-294.

Context. Pediatric intensive care units (PICUs) have expanded nationally, yet few studies have examined the potential impact of regionalization and no study has demonstrated whether a relationship between patient volume and outcome exists in these units. Documentation of an inverse relationship between volume and outcome has important implications for regionalization of care. Objectives. This study examines relationships between the volume of patients and other unit characteristics on patient outcomes in PICUs. Specifically, we investigate whether an increase in patient volume improves mortality risk and reduces length of stay. Design and Setting. A prospective multicenter cohort design was used with 16 PICUs. All of the units participated in the Pediatric Critical Care Study Group. Participants. Data were collected on 11 106 consecutive admissions to the 16 units over a 12-month period beginning in January 1993. Main Outcome Measures. Risk-adjusted mortality and length of stay were examined in multivariate analyses. The multivariate models used the Pediatric Risk of Mortality score and other clinical measures as independent variables to risk-adjust for illness severity and case-mix differences. Results. The average patient volume across the 16 PICUs was 863 with a standard deviation of 341. We found significant effects of patient volume on both risk-adjusted mortality and patient length of stay. A 100-patient increase in PICU volume decreased risk-adjusted mortality (adjusted odds ratio: 0.95; 95% confidence interval: 0.91-0.99), and reduced length of stay (incident rate ratio: 0.98; 95% confidence interval: 0.975-0.985). Other PICU characteristics, such as fellowship training program, university hospital affiliation, number of PICU beds, and children's hospital affiliation, had no effect on risk-adjusted mortality or patient length of stay. Conclusions. The volume of patients in PICUs is inversely related to risk-adjusted mortality and patient length of stay. A further understanding of this relationship is needed to develop effective regionalization and referral policies for critically ill children.

High-Versus Low-Threshold Surfactant Retreatment for Neonatal Respiratory Distress Syndrome—Kattwinkel J, Bloom BT, Delmore P, Glick C, Brown D, Lopez S, et al. Pediatrics 2000;106:282-288.

Surfactant therapy has become an effective standard therapy for infants with respiratory distress syndrome (RDS). The first dose may be given either as prophylaxis immediately after delivery, or as rescue after an infant has developed RDS. Second and subsequent doses are currently recommended by the manufacturers to be administered at minimal levels of respiratory support. Purpose. This study compared the relative efficacy of administering second and subsequent doses of Infasurf surfactant at a low threshold ($F_{IO_2} > 30\%$, still requiring endotracheal intubation) versus a high threshold ($F_{IO_2} > 40\%$, mean airway pressure > 7 cm H_2O) of respiratory support. Methods. A total of 2484 neonates received a first dose of surfactant; 1267 reached conventional retreatment criteria and were randomized to be retreated according to low- or high-threshold criteria. They were then retreated at a minimum of 6-hour intervals each time they reached their assigned threshold until receiving a maximum of 4 total doses. Subjects were stratified by whether they received their first dose by prophylaxis or rescue and by whether their lung disease was considered complicated (evidence of perinatal compromise or sepsis) or uncomplicated. Results. Among the patients randomized, 33% of prophylaxis and 23% of rescue subjects met criteria for the complicated stratum. Although infants allocated to the high-threshold strategy were receiving slightly more oxygen at 72 hours, there was no difference in the number receiving mechanical ventilation at 72 hours or in the secondary respiratory outcomes (requirement for supplemental oxygen or mechanical ventilation at 28 days, supplemental oxygen at 36 weeks' postconceptional age, inspired oxygen concentration >60% at any time). However, there was a significantly higher mortality for infants with complicated RDS who had received retreatment according to the high-threshold strategy. Conclusions. We conclude that equal efficacy can be realized by delaying surfactant retreatment of infants with uncomplicated RDS until they have reached a higher level of respiratory support than is the current standard. We speculate that this would result in a substantial cost-saving from less utilization of drug. Conversely, we believe that infants with complicated RDS should continue to be treated by the low-threshold retreatment strategy, which is currently recommended by the manufacturers of the commercially available surfactants.

Risk Factors for the Development of Hemodynamically Significant Cardiac Arrhythmias in Patients with Mechanical Ventilation—Koh Y, Kim TH, Lim CM, Kim W, Kim YH, Kim DS, Kim WD. J Crit Care 2000;15:46-51.

PURPOSE: The purpose of this article was to identify the risk factors related to development of hemodynamically significant cardiac arrhythmias in patients with mechanical ventilation. MATERIALS AND METHODS: Holter recording and echocardiogram were performed within 24 hours of ventilator initiation in patients on mechanical ventilation (MV) owing to respiratory failure (RF) from various reasons. RESULTS: From 68 patients, hemodynamically significant cardiac arrhythmias were detected in 18 patients (26.5%). Initial mean arterial pressure, maximal heart rate, and initial pH were identified as risk factors for hemodynamically significant cardiac arrhythmias. Additionally, the patients with pressure-controlled ventilation as an initial ventilatory mode developed hemodynamically significant cardiac arrhythmias less frequently than the patients with other modes (15.8% vs. 40%, $p = 0.03$). In multivariate analysis, initial mean arterial pressure (< 70 mm Hg, odds ratio [OR]: 5.5; 95% confidence interval [CI]: 1.2 to 24.2, $p = 0.026$), maximal heart rate (> 120/min, OR: 19.7; 95% CI: 2.0 to 190.9, $p = 0.01$), and pressure-controlled ventilation (OR: 0.13; 95% CI: 0.03 to 0.55, $p = 0.006$) were associated with the development of hemodynamically significant cardiac arrhythmias. CONCLUSIONS: These findings suggest that during the early stages of mechanical ventilation with acute respiratory failure, hemodynamically significant cardiac arrhythmias are directly associated with tachycardia (≥ 120/min), initial MAP (≤ 70 mm Hg), and, inversely, the initial use of pressure-controlled ventilation.

Late Steroid Therapy in Primary Acute Lung Injury—Varpula T, Pettila V, Rintala E, Takkunen O, Valtonen V. Intensive Care Med 2000;26:526-531.

OBJECTIVE: To investigate the effect of steroid treatment in the late phase of primary acute lung injury (ALI) with special emphasis on pneumococcal pneumonia. DESIGN: Retrospective study. SETTING: Multidisciplinary intensive care unit (ICU) in a university hospital. PATIENTS: Of 31 patients with primary ALI requiring mechanical ventilation for more than 10 days, 16 were treated with methylprednisolone and 15 served as controls. MEASUREMENTS AND RESULTS: Steroid and control groups were comparable regarding demographic data, APACHE II score, Multiple Organ Dysfunction Score (MODS), and P_{aO_2}/F_{IO_2}-ratio on admission to ICU. The mean start of steroid therapy was 9.7 days after establishment of respiratory failure, and values for control patients were registered on day 10. The P_{aO_2}/F_{IO_2} ratio improved significantly within 3 days after the start of steroid therapy, and MODS and C-reactive protein decreased concurrently. No differences in mortality, in length of ICU stay, or in length of mechanical ventilation were detectable. In a subgroup analysis, for patients with *Streptococcus pneumoniae* pneumonia,

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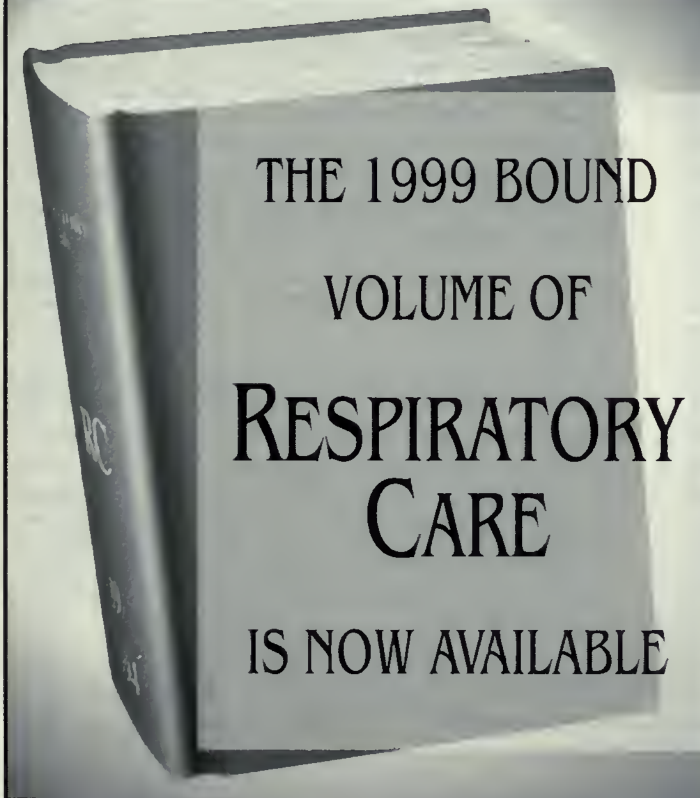

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beneficial change in physiological variables was evident. CONCLUSIONS: In patients with primary ALI, steroid therapy, started 10 days after the start of mechanical ventilation, improves gas exchange and is associated with a decrease in multiorgan dysfunction.

Bioelectrical Impedance Analysis in Estimating Nutritional Status and Outcome of Patients with Chronic Obstructive Pulmonary Disease and Acute Respiratory Failure—Faisy C, Rabbat A, Kouchakji B, Laaban JP. Intensive Care Med 2000;26:518-525.

OBJECTIVE: To evaluate bioelectrical impedance analysis (BIA) in estimating the nutritional status and outcome of patients with chronic obstructive pulmonary disease (COPD) and acute respiratory failure (ARF) in comparison with measurements of anthropometric parameters and plasma levels of visceral proteins. DESIGN: Retrospective study. SETTING: A ten-bed intensive care unit (ICU) in a university teaching hospital. PATIENTS: 51 COPD patients with ARF in whom BIA data, anthropometric parameters, and measurements of visceral proteins were available. MEASUREMENTS AND RESULTS: BIA results in patients requiring mechanical ventilation (MV) vs. those who did not showed lower active cell mass (ACM; 37.5 ± 6.5% vs. 42.4 ± 7.2% body weight, $p = 0.01$) and a higher extra-/intracellular water volume ratio (ECW/ICW; 1.25 ± 0.2 vs. 1.04 ± 0.2, $p = 0.0001$), suggesting a more severe alteration in the nutritional status among those on MV. Anthropometric data showed the opposite results, since body weight, body mass index (BMI), triceps skinfold thickness (TSF), and fat mass were significantly higher in the invasively ventilated patients, whereas middle-arm muscle circumference (MAMC) did not differ between the two groups. The marked inflation of the extracellular compartment (ECW, ECW/ICW) that was well shown by BIA in the invasively ventilated patients presumably lead to inaccurate anthropometric results (overestimation of TSF and fat mass, and erroneous measure of MAMC). A higher death rate (38% vs. 0%, $p = 0.01$) was observed in the patients with ACM depletion (ACM $\leq$ 40.6% body weight, $n = 26$) than in those without ACM depletion ($n = 25$). Low albumin level (< 30 g/L) was associated with increased mortality (33% vs. 7%, $p = 0.04$), but the differences in the other biological and anthropometric parameters (prealbumin and transferrin levels, body weight, BMI, TSF, MAMC, fat mass, and fat-free mass) were not associated with mortality. CONCLUSION: This study suggests that the decrease in BIA-derived ACM is a good indication of malnutrition and of poor outcome in COPD patients with ARF.

The Added Value that Increasing Levels of Diagnostic Information Provide in Prognostic Models to Estimate Hospital Mortality for Adult Intensive Care Patients—de Keizer NF, Bonsel GJ, Goldfad C, Rowan KM. Intensive Care Med 2000;26:577-584.

OBJECTIVE: To investigate in a systematic, reproducible way the potential of adding increasing levels of diagnostic information to prognostic models for estimating hospital mortality. DESIGN: Prospective cohort study. SETTING: Thirty UK intensive care units (ICUs) participating in the ICNARC Case Mix Programme. PATIENTS: Eight thousand fifty-seven admissions to UK ICUs. MEASUREMENTS AND RESULTS: Logistic regression analysis incorporating APACHE II score, admission type and increasing levels of diagnostic information was used to develop models to estimate hospital mortality for intensive care patients. The 53 UK APACHE II diagnostic categories were substituted with data from a hierarchical, five-tiered (type of condition required surgery or not, body system, anatomical site, physiological/pathological process, condition) coding method, the ICNARC Coding Method. The inter-rater reliability using the ICNARC Coding Method to code reasons for admission was good (kappa = 0.70). All new models had good discrimination (AUC = 0.79-0.81) and similar or better calibration compared with the UK APACHE II model (Hosmer-Lemeshow goodness-of-fit $H = 18.03$ to $H = 26.77$ for new models versus $H = 63.51$ for UK APACHE II model). CONCLUSION: The UK APACHE II model can be simplified by extending the admission type and substituting the 53 UK APACHE II diagnostic categories with nine body systems, without losing discriminative power or calibration.

Effect of Hypothermia on Ventilation in Anesthetized, Spontaneously Breathing Rats: Theoretical Implications for Mechanical Ventilation—Torbati D, Camacho MT, Raszynski A, Sussmane JB, Totapally BR, Hultquist K, Wolfsdorf J. Intensive Care Med 2000;26:585-591.

OBJECTIVE: To test if hypothermia, induced by a sustained pentobarbital anesthesia, in rats can reduce ventilatory demands without compromising pulmonary gas-exchange efficiency. DESIGN: Prospective study. SETTING: Research laboratory in a hospital. SUBJECTS: One group of 11 female Sprague Dawley rats. INTERVENTIONS: The rats were anesthetized with 45 mg/kg pentobarbital, tracheostomized and intubated; their femoral veins and arteries were cannulated. After surgery, anesthesia and fluid balance were maintained (10 mg/kg per h pentobarbital, and 5 mL/kg per h saline, i.v.). Rectal temperature, mean arterial blood pressure (MAP), and heart rate (HR) were continuously monitored. The respiratory variables and gas-exchange profiles were determined at 38 degrees C (normothermia), and during stepwise hypothermia at 37, 35, 33, 31 and 29 degrees C. The arterial pressure of carbon dioxide (P_{aCO_2}), pH and arterial pressure of oxygen (P_{aO_2}) during hypothermia were corrected at body temperature. MEASUREMENTS AND RESULTS: Graded systemic hypothermia, with maintained anesthesia, produced a strong correlation between reduction in the respiratory frequency and rectal temperature ($r^2 = 0.55$; $p < 0.0001$; $n = 66$). The minute volume was significantly reduced, starting at 35 degrees C, without significant changes in the tidal volume (repeated measures of analyses of variance followed by Dunnett multiple comparisons test). No significant changes occurred in the P_{aCO_2}, pH, P_{aO_2}, hemoglobin oxygen saturation, the calculated arterial oxygen content and estimated alveolar-arterial oxygen difference during mild hypothermia (37-33 degrees C). The P_{aO_2}, however, was significantly reduced below 31 degrees C. The MAP remained stable at different levels of hypothermia, whereas HR was significantly reduced below 33 degrees C. CONCLUSIONS: Mild hypothermia in rats, induced by a sustained pentobarbital anesthesia, reduces ventilation without compromising arterial oxygenation or acid-base balance, as measured at body temperature. Theoretically, our observations in spontaneously breathing rats imply that a combination of mild hypothermia with anesthesia could be safely utilized to maintain adequate ventilation, using relatively low minute ventilation. We speculate that such a maneuver, if applied during mechanical ventilation, may prevent secondary pulmonary damage by allowing the use of lower ventilator volume-pressure settings.

Dialysis Disequilibrium Syndrome: An Unusual Cause of Respiratory Failure in the Medical Intensive Care Unit—DiFresco V, Landman M, Jaber BL, White AC. Intensive Care Med 2000;26:628-630.

We describe a case of the dialysis disequilibrium syndrome (DDS) that was marked by the rapid onset of cerebral edema and the subsequent development of acute respiratory failure. The patient was treated successfully with a combination of mechanical hyperventilation and mannitol. The clinical presentation, pathogenesis, prevention and treatment of the entity are discussed.

What's That Sound?

This issue of the Journal is very special, since it contains the recently released, revised, clinical practice guideline entitled "Treating Tobacco Use and Dependence." It has been several years since the first guideline was published by the United States Department of Health and Human Services Public Health Service. Since that time, a great deal has been learned regarding all aspects of treating persons who are dependent on tobacco products. This latest version of the guideline draws from contemporary science surrounding perhaps the most predominant factor influencing health care costs, morbidity, mortality, and quality of life. As such, respiratory therapists and all others who provide diagnosis, treatment, and management of patients and consumers who have pulmonary diseases should become thoroughly familiar with the guideline and seize this incredible opportunity to add value to their efforts.

Our nation's health care delivery system is still unable to adequately control costs of health care. Indeed, the health reform movement is far from finished even though reform efforts have been underway for approximately 10 years. Our goal of balancing health care resource with health care need still eludes us. Many providers have undergone and continue to undergo extraordinary financial pressure brought about by decreased reimbursement rates. Many of you have observed negative clinical consequences because of the scarcity of qualified human resources. I believe that the challenge we face will not be successfully addressed until we learn to devote much more time, energy, and treasure to lessening the demand of health care services. When this is accomplished, we'll then be able to allocate resources based on medical necessity rather than by reimbursement policy.

Few would disagree that if we as a society can avoid behaviors that pose health risks we can eventually realize a decrease in health care resource consumption and the associated costs. Among health risk behaviors, tobacco use has been identified as the primary avoidable cause of illness and over 430,000 deaths each year in the United States.[1]

Smoking has been scientifically linked as a cause of chronic obstructive pulmonary disease, heart disease, cancer, stroke, and complications of pregnancy.[2]

Tobacco use translated into more than $50 billion of medical care costs in 1993. Add to that number another $47 billion of nonmedical-related costs as a result of forfeited earnings and lost productivity.[3,4] Now imagine what the system could do with even half of that money if it did not have to be spent to support the effects of disease brought about by tobacco use.

What about the people who consume tobacco products? Even though approximately 25% of adult Americans smoke,[5] 70% want to quit smoking completely, and 46% try to quit each year.[6] The problem is they need help to get the job done. That is where you come in.

All components of the health care delivery system, especially health care services providers, are being challenged to demonstrate their value. We as a profession have done an excellent job of that, especially with regard to the use of protocols.[7,8] Some of you have embraced smoking cessation programs for several years, but have found difficulty in gaining reimbursement. We must now capitalize on the continuing trend to recognize the costs associated with tobacco use and, on the flip side, the savings that can be achieved if tobacco use is managed downward through intervention. Seventy percent of smokers visit a health care setting on at least an annual basis.[9–11] This presents members of the health care delivery team with a tremendous opportunity to intervene and assist persons who are dependent on tobacco products to attain their goal of quitting tobacco use. The guideline is focused especially on persons who have frequent contact with smokers, but doesn't exclude anyone, including insurers and administrators.

Given the diseases and conditions encountered, respiratory therapists are very well positioned to undertake smoking cessation programs, along with other initiatives that lead to decreasing the number of persons using tobacco products in the United States. Given the challenges manifest in the current health care system reform movement, direct involvement by respiratory therapists in smoking cessation programs is not only the right thing to do on behalf of our patients, but it is also the smart thing to do because we can form a better linkage with interventions that lead to decreasing the cost of health care. To me, that spells value-added.

It is disturbing to note that the guideline, even though it concludes that smoking is a highly significant health threat, also states that there exists a "disinclination among clinicians to intervene consistently." This provides an enormous opportunity for those who choose to do so. Many of you are becoming involved in disease management and wellness programs. What better strategy could be undertaken than to organize and implement a comprehensive smoking cessation program, and to do so under the umbrella of respiratory care services. There now exist a variety of interventions that have been scientifically evaluated. When these interventions are delivered in a timely and effective manner, the result is a significant reduction of the smoker's risk of acquiring a smoking-related disease.[12,13]

Even though some of you may not be fully aware of which treatments are effective or how the intervention can be provided, don't feel too uncomfortable, since the guideline document indicates that many health care providers are in the same position. That problem can be solved by your reading the guideline thoroughly, and then applying common sense.

Smoking cessation interventions will move forward with or without us. I believe that the sound you hear is opportunity knocking. We like to style ourselves as key players on the health care delivery team. How can we earn that title if we're not involved with the elimination of the chief avoidable cause of illness?

Indeed the worm has turned for health care in this country. Even though we've given a tremendous amount of lip service to disease prevention and health promotion, we still spend much more time and money treating the effects of smoking than avoiding the cause. It will take time to change our thinking as a society, but we as respiratory therapists can help with this endeavor by aggressively organizing and implementing smoking cessation programs in all care settings. The profession has done an excellent job of providing clinical services to patients with pulmonary diseases and problems. We now must grow our efforts on the prevention side and lead by targeting the number one health risk, which, of course, is also the number one pulmonary health risk. Respiratory therapists have a legitimate role to play in the health care delivery system, but that role must expand if we are to realize our potential as health care providers, disease state managers, consumer educators, and wellness promoters. I urge all of you to thoroughly acquaint yourselves with the guideline and undertake efforts to act on it. It's a good fit regardless of care setting. Remember, it's the smart thing to do, but it's also the right thing to do. Opportunity is knocking!

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Pressure Support and Pressure Assist/Control: Are There Differences? An Evaluation of the Newest Intensive Care Unit Ventilators

Purris Williams RRT, Matt Muelver, Joe Kratohvil RRT, Ray Ritz RRT FAARC,
Dean R Hess PhD RRT FAARC, and Robert M Kacmarek PhD RRT FAARC

BACKGROUND: Pressure support (PS) has been widely studied in both patients and lung models, but there is little data available evaluating pressure assist/control (P A/C, frequently referred to as PCV) and no data comparing the operational capabilities of these two modes on the newest generation of ICU ventilators. We used a spontaneously breathing lung model to evaluate the response of the following new generation ventilators to varying inspiratory demand in both PS and P A/C: BEAR 1000, Dräger Evita 4, Hamilton Galileo, Nellcor Puritan-Bennett 840 and 740, Siemens Servo 300A, TBird AVS. METHODS: A bellows-in-a-box lung model was set at a respiratory rate of 12 breaths/min, inspiratory time of 1.0 second, and peak inspiratory flows (modified square wave) of 40, 60, and 80 L/min. Each ventilator was set at three levels of PS and P A/C: 10, 15, and 20 cm H_2O. On all ventilators, flow-triggering was set as sensitive as possible without causing self-triggering. RESULTS: Trigger pressure, trigger pressure-time product, inspiratory trigger time delay, ventilator-delivered peak flow, inspiratory area as a percent of the ideal inspiratory area, expiratory time delay, supraplateau expiratory pressure change, and expiratory area all varied among ventilators and at different lung model peak flows ($p < 0.01$ and $\geq 10\%$ difference). However, PS and P A/C on a given ventilator only differed with regard to expiratory variables ($p < 0.01$ and $\geq 10\%$ difference). CONCLUSION: In a given ventilator little difference exists in gas delivery and response variables between PS and P A/C, but performance differences do exist among the ventilators evaluated. Ventilator performance is diminished at high lung model peak flows and low pressure settings. *Key words: pressure support, pressure assist, pressure control, ventilator triggering, expiratory retardation, expiratory delay, delivered peak flow.* [Respir Care 2000;45(10):1169–1181]

Background

Over the last 15 years, much emphasis has been placed on the use of pressure targeted ventilation.[1–10] Pressure support ventilation (PS) has been extensively studied in patients[1–8] and in lung models,[9–10] and is considered advantageous by many because of its ability to vary delivered flow based on patient demand.[11–13] Pressure control (PC or PCV) has been advocated for the management of acute respiratory distress syndrome patients[14–17] because of its ability to limit peak alveolar pressure and its exponentially decelerating flow pattern. The term assist/control (A/C) has been almost exclusively used in reference to the application of volume assisted ventilation, with little reference to pressure A/C (P A/C).[18] As with volume control, patients receiving pressure control can trigger the ventilator with appropriate setting of the trigger sensitivity and, as with PS, peak airway pressure is targeted while ventilator-delivered flow varies with demand of the patient.[19] The practitioner clearly has two options for pressure ventilation. Pressure A/C can theoretically be used in the same circumstance in which volume A/C is employed. Its advantages over PS include a backup rate and a set inspiratory time (T_I), whereas PS gives control over ending inspiration to the patient. What has not been clearly defined is the gas delivery and ventilator response differences, if

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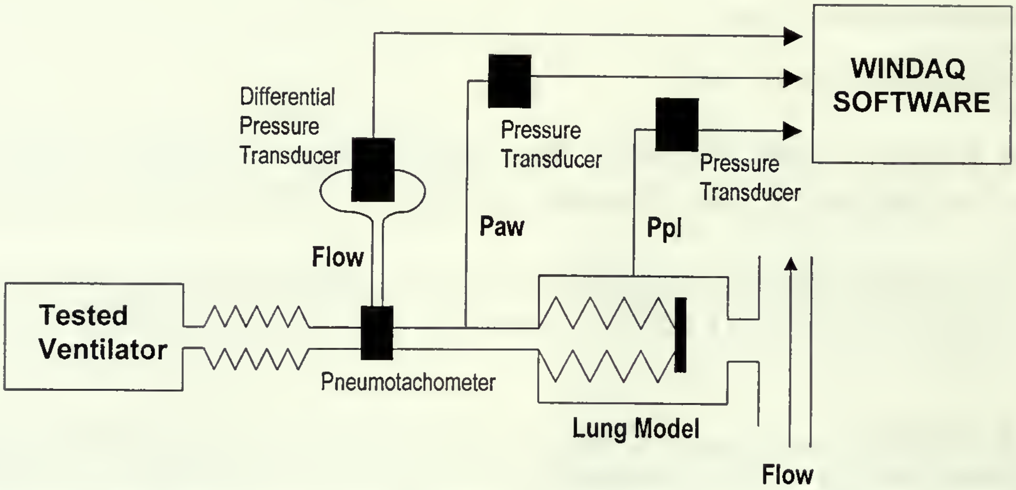

Fig. 1. Diagram of the experimental setup. P_{aw} = airway pressure. P_{pl} = simulated pleural pressure of the lung model. See text for description.

any, between these two (PS and P A/C) pressure targeted assist modes.

Most new generation intensive care unit (ICU) ventilators provide both pressure support (PS) and pressure assist/control (P A/C) ventilation.[19,20] The specific operational difference between these two modes is the mechanism that transitions inspiration to expiration. With pressure support the primary mechanism is a decrease in peak inspiratory flow to a predetermined level, whereas with P A/C mechanical T_I is preset.[19,20]

We compared the operation of seven of the newest generation ICU ventilators in a spontaneously breathing lung model in both PS and P A/C. We hypothesized that there would be no difference in variables assessed between PS and P A/C except for the transition to expiration and that there would be no difference in response among ventilators evaluated.

Materials and Methods

Lung Model

A bellows-in-a-box lung model was used to simulate spontaneous breathing (Fig. 1). The space between the rigid box and the bellows functioned similar to the pleural space. The box was connected to a rigid T-tube of one-eighth inch internal diameter through which a gas flow was introduced to create negative pressure in the pleural space by the jet drag effect. Source gas (50 psi, oxygen) was connected to a pressure regulator (SMC AR200 [7–120 psi], SMC Company, Tokyo, Japan) and a proportional solenoid valve (SMC 315, SMC Company, Tokyo, Japan). A function generator (EGC 2230, Kenwood, Tokyo, Japan) controlled the opening of the solenoid valve. Adjustment of the regulator varied flow through the T-tube, allowing variation of the negative pressure in the pleural space, and the function generator simulated a modified square wave flow pattern. Thus, inspiratory flow demand, respiratory rate, and T_I were controlled independently. A linear resister (7100 R-5, Hans Rudolph, Kansas City, Missouri) created an airway resistance of 8.2 cm H_2O/L/s at a flow of 60 L/min. Compliance of the lung model (50 mL/cm H_2O) was determined by spring settings on the bellows. For this study the lung model was set as follows: spontaneous breathing, respiratory rate of 12 breaths/min, T_I of 1.0 second, modified square wave flow pattern and peak inspiratory flows of 40, 60, and 80 L/min. This flow pattern was selected because it most closely resembled the pattern in highly stressed patients.

Ventilators Compared

Seven critical care ventilators were evaluated: (1) Bear 1000 (Thermo Respiratory Group, Palm Springs, California), (2) Dräger Evita 4 (Dräger, Telford, Pennsylvania), (3) Hamilton Galileo (Hamilton Medical, Rhäzüns, Switzerland), (4) Puritan-Bennett 840 (Mallinckrodt, Pleasanton, California), (5) Puritan-Bennett 740 (Mallinckrodt, Pleasanton, California), (6) Siemens Servo 300A (Siemens-Elema, Solna, Sweden), and (7) TBird AVS (Thermo Respiratory Group, Palm Springs, California). Each ventilator was connected to the lung model using a standard heated wire circuit (Hudson Respiratory Care, Temecula, California) and a Conchatherm IV humidifier (Hudson Respiratory Care, Temecula, California) (Table 1). The humidification system, however, was not activated during the study, in order to avoid water accumulation in the lung model.

Table 1. Specific Settings of Studied Ventilators

Ventilator	Modes	Inspiratory Sensitivity	Pressure Rise Time	Expiratory Sensitivity
BEAR 1000	P A/C, PS	Sens 1.0 L/min Bias flow 5 L/min	0 (midpoint)	Preset at 30%
Dräger Evita 4	P A/C, PS	0.3 L/min	0.0 s (fastest)	Preset at 25%
Hamilton Galileo	P A/C, PS	2.0 L/min	50 ms (fastest)	25%
Puritan-Bennett 740	PS	1 L/min	Preset	Preset at 25%
Puritan-Bennett 840	P A/C, PS	0.5 L/min (Base flow 2 L/min)	100% (fastest)	25%
Siemens Servo 300A	P A/C, PS	Green/red line (Bias flow 2 L/min)	0% (fastest)	Preset at 5%
TBird AVS	P A/C, PS	Sens 1 L/min (Bias flow 10 L/min)	Preset	Preset at 25%

P A/C = pressure assist/control. PS = pressure support.

Measurement and Calibration

A pneumotachometer (model 3700A, Hans-Rudolph, Kansas City, Missouri) was placed at the airway opening of the lung model (see Fig. 1). The pressure differential across the pneumotachometer was measured (45–14-871 [± 2 cm H_2O], Validyne; Northridge, California), amplified (model 8805C, Hewlett Packard, Waltham, Massachusetts), digitized, and converted to a flow signal using a computerized graphics program (WINDAQ, Dataq Instruments, Akron, Ohio). The pneumotachograph was calibrated at 1 L/s flow delivered by a precision flow meter (Brooks Instruments, Hatfield, Pennsylvania). Pressures at the airway opening and in the simulated pleural space were measured with differential pressure transducers (45–32-871 [± 100 cm H_2O], Validyne). All pressure transducers were zeroed and calibrated at 20 cm H_2O using a water manometer. Pressure signals were amplified (8805 C, Hewlett Packard, Waltham, Massachusetts) and digitized using WINDAQ.

Data Acquisition and Analysis

All signals were digitized at 100 Hz and recorded using WINDAQ. The WINDAQ playback software was used to analyze data. Three breaths were analyzed for each experimental setting.

Measurements Recorded during the Inspiratory Phase

The beginning and end of the inspiratory phase were determined from changes in the simulated pleural pressure (Fig. 2). A negative deflection in pleural pressure indicated the start of the inspiratory phase and the subsequent positive deflection signaled the end of inspiration. From the beginning of inspiration, the time required to decrease airway pressure to the lowest pressure below baseline was recorded as the inspiratory trigger delay time (D_T). The time interval between the lowest pressure below baseline and the return to baseline pressure was defined as inspiratory baseline delay time (D_B). The sum of D_T and D_B, the time from the beginning of inspiration to the return of airway pressure to baseline, was defined as the total inspiratory delay time (D_{TOT}). Inspiratory trigger pressure (P_T) was defined as the difference between baseline pressure (positive end-expiratory pressure or PEEP) and the lowest pressure below baseline during triggering of inspiration. The trigger pressure-time product (T-PTP) was defined as the area of the airway pressure (P_{aw})-time tracing below baseline pressure (PEEP) during triggering. The peak flow delivered by the ventilator was recorded. The area of the P_{aw}-time tracing above and below baseline during the inspiratory phase was calculated and expressed as a percentage (inspiratory area percent, Area I%) of the ideal inspiratory area. The area of the rectangle created by the difference between peak inspiratory pressure (PIP) and baseline pressure (PEEP), and T_t as defined by the pleural pressure tracing (see Fig. 2) was considered the ideal inspiratory area. The actual total Area I was equal to Area I plus T-PTP (see Fig. 2).

Measurements Recorded during the Expiratory Phase

Expiratory delay time (D_E) was defined as the time between the end of inspiration as determined by change in pleural pressure and the point where P_{aw} returned to the

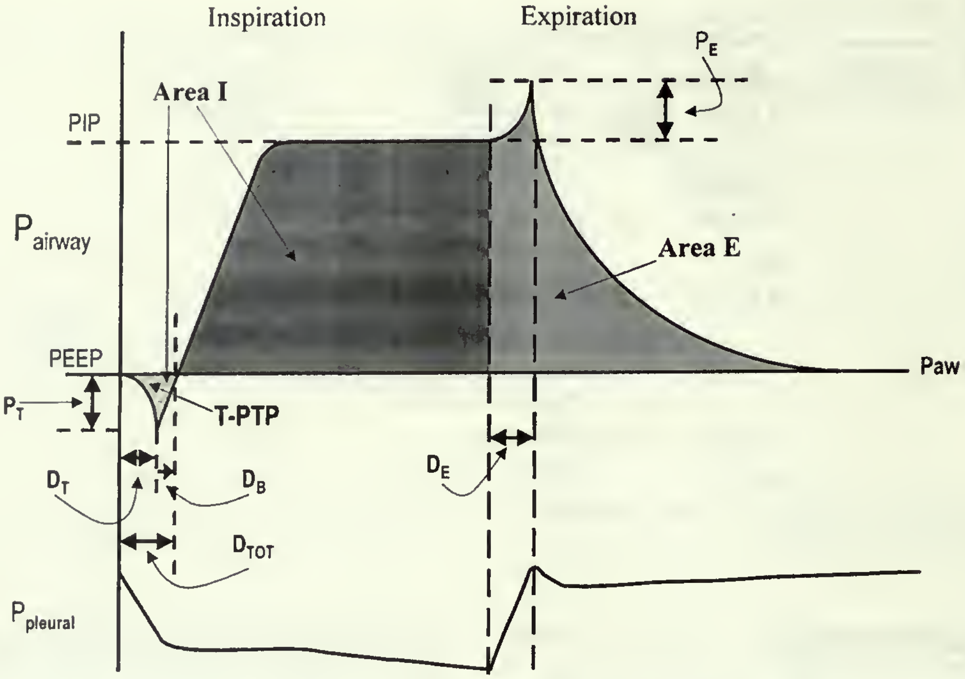

Fig. 2. Simulated pleural and airway pressure curves illustrating the variables evaluated during the inspiratory and expiratory phases. PIP = peak inspiratory pressure. PEEP = positive end-expiratory pressure (baseline pressure). P_T = inspiratory trigger pressure. D_T = inspiratory trigger delay time. T-PTP = trigger pressure-time product. D_B = inspiratory baseline delay time . D_{TOT} = total inspiratory delay time. D_E = expiratory delay time. P_{aw} = airway pressure. P_E = supraplateau expiratory pressure change.

end-inspiratory level (see Fig. 2). Supraplateau expiratory pressure change (P_E) was defined as the pressure change above PIP following the beginning of exhalation. The rate at which pressure decreased to baseline after the beginning of exhalation was expressed as the area of the P_{aw}-time tracing during the expiratory phase (Area E).

Experimental Protocol

All of the ventilators (except the Puritan-Bennett 740) were set to provide 5 cm H_2O of PEEP and three levels (10, 15, and 20 cm H_2O) of pressure support (PS) and pressure assist/control (P A/C) corresponding to identical PIPs of 15, 20 and 25 cm H_2O. During P A/C, T_I was set at 1.0 second and backup rate at 8/min. Flow-triggering was set on all ventilators to be as sensitive as possible without causing self-triggering. When applicable, inspiratory pressure rise time was set to the most rapid (fastest) setting. On those ventilators with variable inspiratory termination criteria, it was set at 25% of peak inspiratory flow (Table 1). The Puritan-Bennett 740 was only studied in PS, as P A/C is not available from the 740.

Statistical Analysis

All values are reported as mean ± SD. The following dependent variables were evaluated: D_T, D_B, D_{TOT}, P_T, T-PTP, peak flow, Area I%, Area E, P_E, and D_E. Each dependent variable was evaluated using a one-way analysis of variance (ANOVA) for each independent variable (ventilator, PC or PS level, and lung model inspiratory flow). A two-way ANOVA was used to evaluate relationships between independent variables. Post hoc analysis was performed with the Scheffé test. Statistical significance was defined as $p < 0.01$. Data are only reported when, in addition to statistical significance, a ≥ 10% difference existed between variables. This level of significance was selected because of the very small standard deviations observed with lung model studies resulting in differences equal to measurement errors being significant if $p < 0.05$ was used.

Results

Waveforms

Representative (filtered) waveforms for pressure versus time and flow versus time during PS and P A/C are shown in Figures 3 and 4. During inspiration, the waveforms for a given ventilator showed little difference between PS and P A/C, indicating similar performance characteristics regardless of the mode of ventilation. However, during the expiratory phase, differences between modes were identified for most variables evaluated. The pressure above the

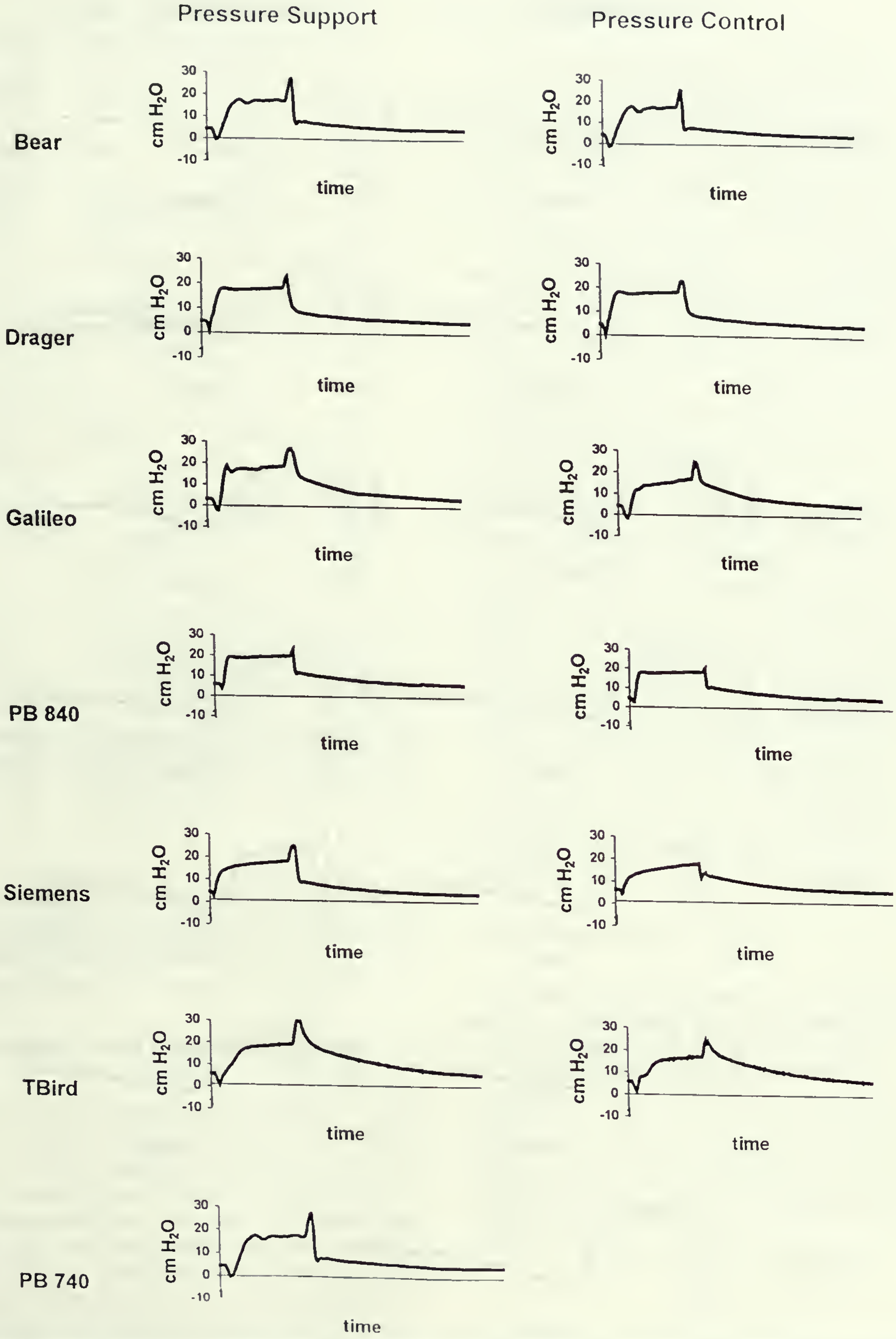

Fig. 3. Filtered pressure versus time tracings for each ventilator in pressure support and pressure control at settings of 15 cm H_2O, lung model peak flow of 60 L/min, and lung model inspiratory time of 1 second.

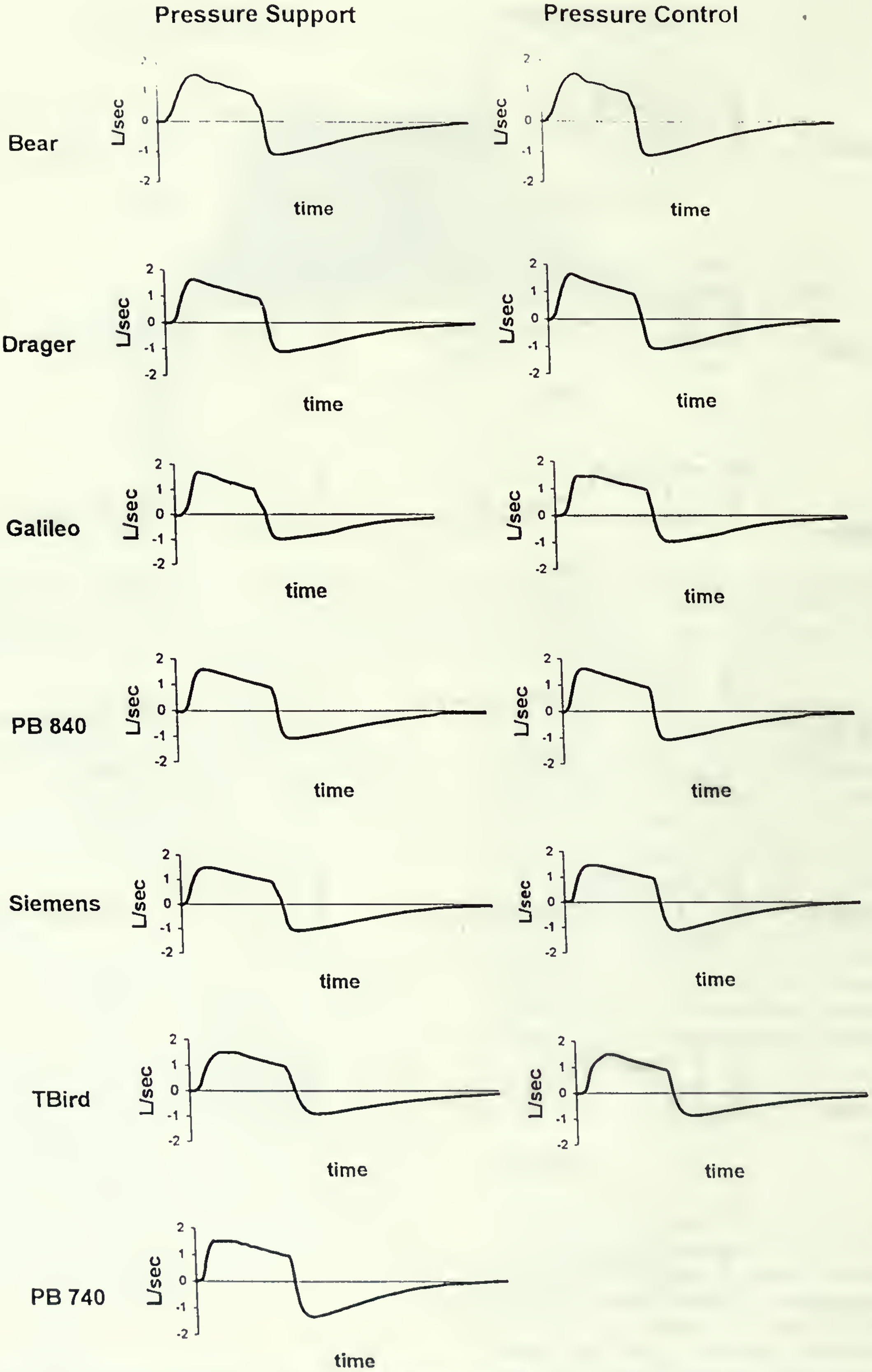

Fig. 4. Filtered flow versus time tracings for each ventilator in pressure support and pressure control at settings of 15 cm H_2O, lung model peak flow of 60 L/min, and lung model inspiratory time of 1 second.

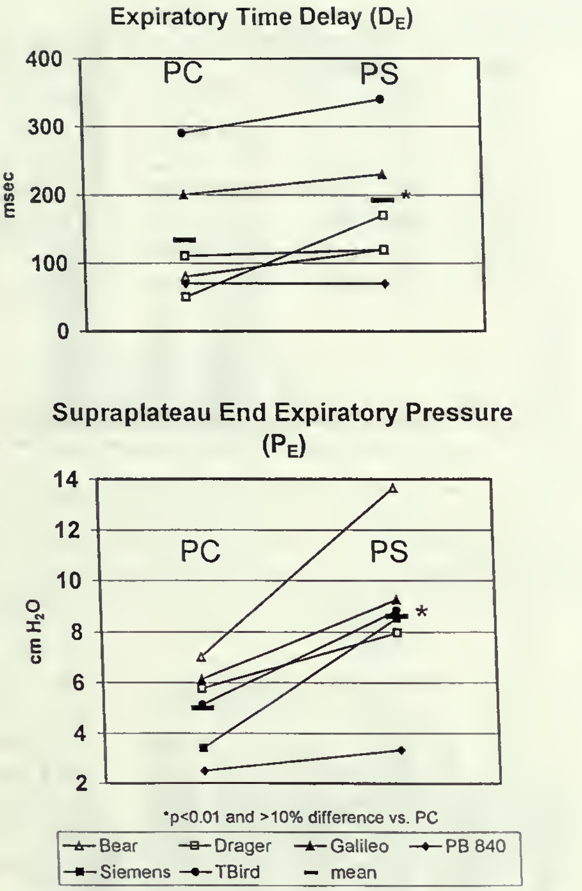

Fig. 5. Supraplateau expiratory pressures and expiratory delay times during pressure support (PS) and pressure control (PC) across all lung model and ventilator settings.

inspiratory plateau is noticeably greater during PS for all ventilators evaluated. The expiratory time delay was also greater during PS than during P A/C. Flow waveforms for all ventilators appeared to be similar in both modes.

Pressure Support versus Pressure Assist/Control

Individual ventilator mean values for supraplateau end-expiratory pressure and T_E delay averaged across all experimental settings during PS and P A/C are shown in Figure 5. For both variables, values for P A/C were consistently lower than PS ($p < 0.01$ and $\geq 10\%$ differences). This was not true for other variables evaluated, as shown in Tables 2 and 3, which list composite data across all experimental settings in PS and P A/C. Although minor, sometimes significant differences do exist; in the vast majority of settings the differences were not $> 10\%$. For this reason, additional data will only be presented for PS.

Effect of Peak Flow

As noted in Figures 6 through 10 and Table 4, the most striking differences between variables evaluated occurred when lung model peak inspiratory flow was altered. Large differences in trigger pressure at specific lung model peak flows were observed among ventilators (see Fig. 6) as well as differences in the T-PTP ($p < 0.01$ plus $\geq 10\%$) (see Fig. 7). Both increased as lung model flow increased and both differed among ventilators. With most ventilators, there were differences in percent inspiratory area ($p < 0.01$ plus $\geq 10\%$) (see Fig. 8) as peak lung model flow increased and at each lung model flow among ventilators. Inspiratory areas decreased as peak lung model flow increased. Supraplateau expiratory pressure (see Fig. 9) varied greatly among ventilators as well as among peak lung model flow setting ($p < 0.01$ plus 10%). Increasing peak lung model flow resulted in greater supraplateau end-inspiratory pressure. Expiratory area (see Fig. 10) also varied among ventilators and among lung model peak flows for each ventilator ($p > 0.01$ plus 10%). Again, the greater the lung model peak flow, the greater the expiratory area. Trigger delay times, ventilator-delivered peak flows, and expiratory delay times at all lung model peak flows are listed in Table 4. Trigger delay time was short and variably affected by increasing lung model peak flow, but did differ among ventilators ($p < 0.01$ plus 10%). Inspiratory baseline delay time, total inspiratory delay time, and expiratory delay time were increased as peak lung model flow increased and did differ among ventilators ($p < 0.01$ plus 10%). Ventilator delivered peak flows in some ventilators also increased as lung model peak flow increased, and differences did exist among some ventilators ($p < 0.01$ plus 10%).

Effect of Set Pressure

The effects of PS level averaged across all lung model peak flows are listed in Table 5. As noted, there are differences among ventilators and between pressure settings ($p < 0.01$ plus 10%), but in general the magnitude of the differences are not as large as those observed with alterations in lung model peak flow. For most comparisons, except for expiratory area, individual ventilators varied least from ideal function at higher pressure settings. Expiratory area was lowest in every ventilator at the lowest pressure settings.

Discussion

The primary findings of this study can be summarized as follows:

Table 2. Average Ventilator Performance Across All Settings in Pressure Assist/Control

Ventilator	P_T (cm H_2O)	D_T (ms)	D_B (ms)	D_{TOT} (ms)	VPF (L/s)	T-PTP (cm $H_2O \cdot s$)	Area I %	Area E (cm $H_2O \cdot s$)
BEAR	6.26 ± 2.73*†	92 ± 17*†	86 ± 31*†	179 ± 44*†	1.39 ± 0.17	0.67 ± 0.44*†	61.87 ± 11.33*†	5.75 ± 1.28
Dräger	4.39 ± 1.17*†‡	65 ± 15*†‡	46 ± 28*†‡	111 ± 32*†‡	1.44 ± 0.14	0.21 ± 0.12‡	76.56 ± 7.84*	6.35 ± 1.85*†‡
Galileo	5.79 ± 1.68*†	100 ± 6*†	64 ± 12*†	164 ± 15*†	1.38 ± 0.13	0.49 ± 0.18*†	65.13 ± 7.58*†	11.44 ± 0.78*†‡
PB840	1.48 ± 0.46‡	55 ± 10‡	23 ± 4‡	78 ± 8‡	1.53 ± 0.27†	0.05 ± 0.03‡	86.88 ± 2.03‡	5.98 ± 0.97
Siemens	1.92 ± 0.50‡	49 ± 5‡	29 ± 18‡	78 ± 16‡	1.34 ± 0.15*	0.07 ± 0.04‡	74.68 ± 8.69*‡	6.04 ± 1.47
TBird AVS	4.05 ± 1.63*†‡	75 ± 6*†‡	56 ± 37*†‡	131 ± 42*†‡	1.34 ± 0.20*	0.27 ± 0.18‡	69.23 ± 10.26*	13.84 ± 2.68*†‡

All values are means ± SD.

*$p < 0.01$ and >10% difference vs Puritan-Bennett 840.

†$p < 0.01$ and >10% difference vs Siemens.

‡$p < 0.01$ and >10% difference vs BEAR 1000.

P_T = inspiratory trigger pressure. D_T = inspiratory trigger delay time. D_B = inspiratory baseline delay time. D_{TOT} = total inspiratory delay time. VPF = peak flow delivered by the ventilator. T-PTP = trigger pressure-time product. Area I % = area under the airway pressure-time tracing during inspiration expressed as a percentage of the ideal inspiratory area. Area E = area under the airway pressure-time tracing from the beginning of expiration until the return to PEEP. PB = Puritan-Bennett.

Table 3. Average Ventilator Performance Across All Settings in Pressure Support

Ventilator	P_T (cm H_2O)	D_T (ms)	D_B (ms)	D_{TOT} (ms)	VPF (L/s)	T-PTP (cm $H_2O \cdot s$)	Area I %	Area E (cm $H_2O \cdot s$)
BEAR	6.96 ± 2.73*†	94 ± 15†	92.8 ± 29*†	187 ± 41*†	1.43 ± 0.15	0.77 ± 0.46*†	59.94 ± 12.25*†	6.70 ± 1.89
Dräger	4.52 ± 1.40*†‡	62 ± 8	45.5 ± 25	107 ± 25	1.43 ± 0.12	0.22 ± 0.13‡	75.28 ± 7.12*‡	6.79 ± 1.64
Galileo	5.34 ± 1.49*†	95 ± 7*†	52.5 ± 10	147 ± 13*†‡	1.49 ± 0.21	0.39 ± 0.14*†‡	72.14 ± 6.93*‡	12.67 ± 1.28*†‡
PB740	4.41 ± 1.02*†‡	65 ± 12	26.2 ± 6‡	91 ± 9‡	1.41 ± 0.15	0.18 ± 0.06‡	77.00 ± 5.59*‡	4.67 ± 1.59†
PB840	1.49 ± 0.36‡	54 ± 4	22.5 ± 4‡	76 ± 7‡	1.57 ± 0.23†	0.06 ± 0.05‡	86.86 ± 1.62‡	6.25 ± 1.01
Siemens	1.81 ± 0.42‡	48 ± 6	30.7 ± 17‡	79 ± 16‡	1.33 ± 0.15*	0.07 ± 0.04‡	73.49 ± 8.38*‡	7.38 ± 2.31
TBird AVS	4.04 ± 1.50*†‡	79 ± 12	62.5 ± 49*‡	141 ± 57*†‡	1.35 ± 0.20*	0.26 ± 0.15*†‡	69.54 ± 9.11*‡	14.92 ± 3.56*†‡

All values are means ± SD.

*$p < 0.05$ and >10% difference vs Puritan-Bennett 840.

†$p < 0.05$ and >10% difference vs Siemens.

‡$p < 0.05$ and >10% difference vs BEAR 1000.

P_T = inspiratory trigger pressure. D_T = inspiratory trigger delay time. D_B = inspiratory baseline delay time. D_{TOT} = total inspiratory delay time. VPF = peak flow delivered by the ventilator. T-PTP = trigger pressure-time product. Area I % = area under the airway pressure-time tracing during inspiration expressed as a percentage of the ideal inspiratory area. Area E = area under the airway pressure-time tracing from the beginning of expiration until the return to PEEP. PB = Puritan-Bennett.

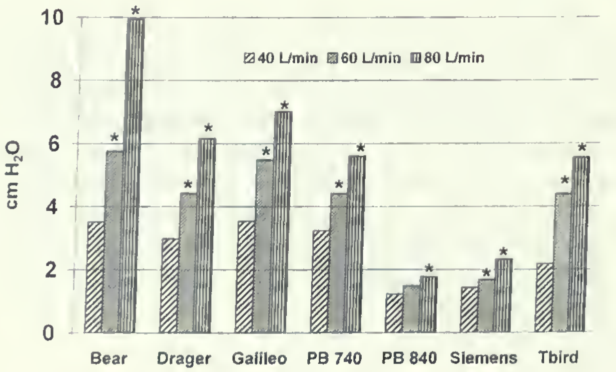

Fig. 6. Maximum sub-baseline trigger pressure (P_T) change across all pressure settings during pressure support (PS) at lung model peak flows of 40, 60, and 80 L/min. *$p < 0.01$ and > 10% difference versus 40 L/min. Differences existed among ventilators at each lung model peak flow ($p < 0.01$ and > 10% difference).

1. Essentially no differences in gas delivery and ventilator response exist between PS and P A/C, but differences do exist in the transition from inspiration to expiration;
2. Large differences exist among the ventilators evaluated in all variables compared;
3. The response of most ventilators diminished as lung model peak flow increased; and
4. Increasing pressure settings resulted in ventilators performing closer to their ideal function.

Pressure Support versus Pressure Assist/Control

Pressure support has been extensively studied as a mode of assisted ventilation,[1-8] whereas pressure assist/control has rarely been evaluated as a mode of assisted ventilation.[18,21] Pressure A/C (pressure control, frequently referred to as PCV, with the sensitivity appropriately set) has primarily been studied as a mode reserved for the management of severely ill acute respiratory distress syndrome

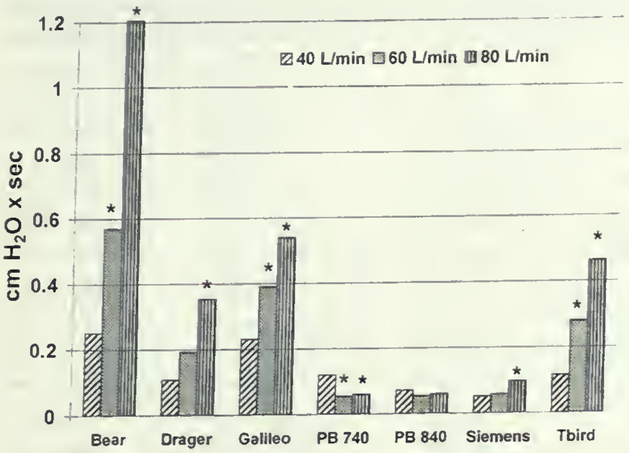

Fig. 7. Trigger pressure-time product (T-PTP) across all pressure settings during pressure support (PS) at lung model peak flows of 40, 60, and 80 L/min. *$p < 0.01$ and > 10% difference versus 40 L/min. Differences existed among ventilators at each lung model peak flow ($p < 0.01$ and > 10% difference).

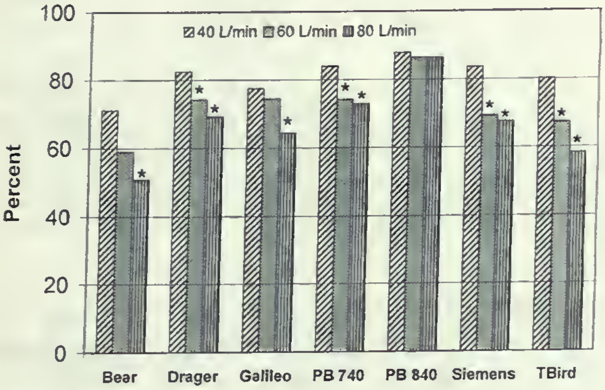

Fig. 8. Percent inspiratory area (% Area I) across all pressure settings during pressure support (PS) at lung model peak flows of 40, 60, and 80 L/min. *$p < 0.01$ and > 10% difference versus 40 L/min. Differences existed among ventilators at each lung model peak flow ($p < 0.01$ and > 10% difference).

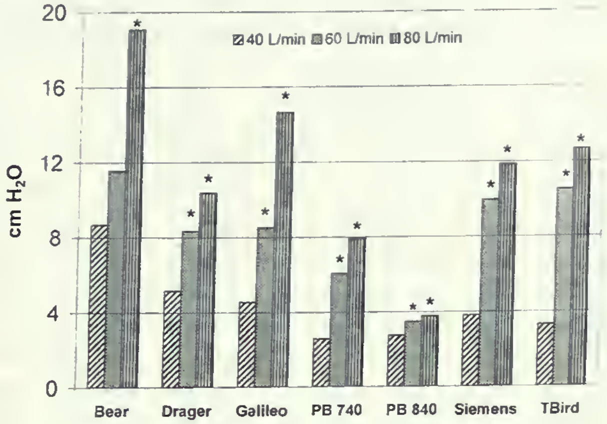

Fig. 9. Supraplateau expiratory pressures (P_E) across all pressure settings during pressure support (PS) at lung model peak flows of 40, 60, and 80 L/min. *$p < 0.01$and > 10% difference versus 40 L/min. Differences existed among ventilators at each lung model peak flow ($p < 0.01$ and > 10% difference).

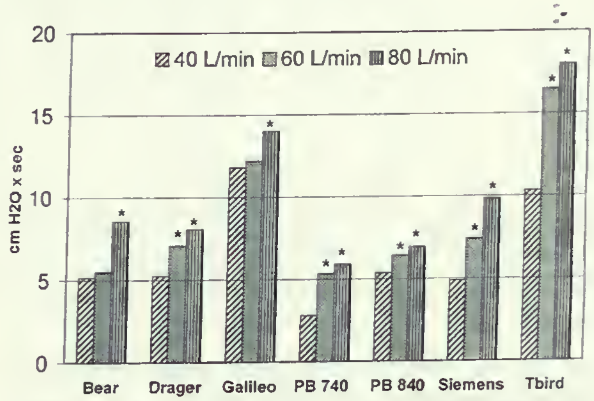

Fig. 10. Expiratory area across (Area E) all pressure settings during pressure support (PS) at lung model peak flows of 40, 60, and 80 L/min. *$p < 0.01$ and > 10% difference versus 40 L/min. Differences existed among ventilators at each lung model peak flow ($p < 0.01$ and > 10% difference).

patients, frequently with the inspiratory-to-expiratory ratio increased.[15–17] The term assist/control has consistently been used to define volume-targeted ventilation, but is rarely used to define the same operational mode with a pressure target (P A/C).[19,20] However, as shown in this comparison, PS and P A/C respond and deliver gas in essentially the same manner. Operationally, the only difference between these two modes is the method of transitioning to exhalation. With PS, exhalation is primarily terminated when delivered flow decreases to a predetermined level. The secondary criteria for inspiratory termination in PS are an increase in pressure above set level (1–20 cm H_2O) and T_I exceeding a maximum preset time (2–5 s).[19,20] During P A/C the primary cycling mechanism is set T_I. However, inspiration is also terminated if airway pressure reaches the clinician-set high pressure limit.[19,20]

The primary differences observed between the operation of PS and P A/C were in those variables derived during expiration. As noted in Figure 5, the supraplateau end-expiratory pressure and the T_E delay were both greater in PS than in P A/C. This difference, however, may have been a result of the experimental protocol and lung model used. The lung model was always set at a T_I of 1 second, as was the T_I during P A/C. With PS the inspiratory termination criteria was either preset (Bear, Dräger, Bird, PB740, Siemens) or set by us (Hamilton, PB840) at 25% of peak flow. Since the lung model flow pattern did not decrease flow to near zero (modified square wave flow) before the end of the inspiratory phase in all settings during PS, the lung model terminated the inspiratory phase before the ventilator identified the end of inspira-

Table 4. Ventilator Performance Averaged for All Pressure Settings During Pressure Support at Lung Model Peak Flows of 40, 60 and 80 L/min

Ventilator	Lung Model Flow Rate	D_T (ms)	D_B (ms)	D_{TOT} (ms)	VPF (L/s)	D_E (ms)
BEAR	40	77 ± 12	63 ± 19	140 ± 24	1.25 ± 0.19	115 ± 5
	60	100 ± 9*	100 ± 23*	200 ± 30*	1.50 ± 0.07*	128 ± 8
	80	101 ± 11*	107.7 ± 25*	208.8 ± 31*	1.49 ± 0.00*	143 ± 9*
Dräger	40	66 ± 5	23 ± 5	89 ± 6	1.32 ± 0.15	96 ± 10
	60	57 ± 9*	44 ± 19*	101 ± 25	1.48 ± 0.12*	116 ± 17
	80	63 ± 9	69 ± 21*	132 ± 17*	1.49 ± 0.14*	142 ± 34*
Galileo	40	91 ± 3	47 ± 11	138 ± 10	1.34 ± 0.15	187 ± 19
	60	100 ± 5*	51 ± 6	151 ± 6	1.70 ± 0.12*	198 ± 30
	80	93 ± 9	60 ± 9*	153 ± 16*	1.44 ± 0.15	307 ± 147*
PB740	40	78 ± 4	21 ± 3	99 ± 6	1.26 ± 0.17	90 ± 17
	60	61 ± 8*	27 ± 5	88 ± 7*	1.47 ± 0.09*	90 ± 22
	80	57 ± 9*	31 ± 6*	88 ± 10*	1.49 ± 0.10*	107 ± 16
PB840	40	52 ± 4	21 ± 3	73 ± 5	1.34 ± 0.12	78 ± 10
	60	54 ± 5	22 ± 4	77 ± 9	1.61 ± 0.09*	72 ± 7
	80	54 ± 5	24 ± 5	79 ± 8	1.77 ± 0.12*	74 ± 5
Siemens	40	51 ± 3	18 ± 4	69 ± 3	1.16 ± 0.15	130 ± 7
	60	48 ± 4	27 ± 9	74 ± 10	1.39 ± 0.08*	153 ± 14
	80	46 ± 7	48 ± 19*	93 ± 20*	1.44 ± 0.12*	214 ± 102*
TBird	40	78 ± 7	30 ± 0	108 ± 7	1.10 ± 0.24	166 ± 28
	60	77 ± 7	63 ± 36	140 ± 40	1.46 ± 0.10*	373 ± 151*
	80	81 ± 20	94 ± 64	176 ± 79	1.49 ± 0.24*	468 ± 168*

All values are mean ± SD.

*$p < 0.01$ and >10% difference vs 40 L/min for each ventilator.

D_T = inspiratory trigger delay time. D_B = inspiratory baseline delay time. D_{TOT} = total inspiratory delay time. VPF = peak flow delivered by the ventilator. D_E = time delay between the end of inspiration and the return of airway pressure to plateau pressure. PB = Puritan-Bennett.

tion. This may have accounted for the greater variations from ideal on all expiratory variables during PS.

Triggering

The primary factor that affected triggering response was lung model peak flow. At high lung model peak flows the triggering response was extended. As has been previously reported by others,[22,23] the subbaseline pressure deflections required to trigger ventilators to inspiration can be divided into two phases, the actual trigger phase depicted by the maximum pressure change (P_T) and the time to reach this pressure (D_T). The second phase D_B, is the time required to return pressure to baseline. D_T and P_T are primarily affected by the drive of the patient, with increased drive resulting in greater P_T in a shorter time period (D_T), and the mechanical response of the ventilator, with poorer response resulting in greater D_T and P_T.[22,23] D_B is affected by the speed with which the ventilator establishes system flow and the level of set pressure.[22,23] Our data indicate that D_B decreased as set pressure increased in most ventilators, some greater than others. This is somewhat explained by the manufacturers' attempts to avoid excessive airway pressure (exceeding set level) from being established early in the inspiratory phase. The higher the set pressure the more aggressive the initial flow can be without overshooting the set target pressure. In those ventilators where little difference in D_B is observed as lung model peak flow is altered, a set time interval between sensing of triggering and obtaining peak pressure is normally programmed. These ventilators can more accurately modulate flow to avoid pressure overshoots at the onset of inspiration. T-PTP is a composite of all of these variables and is also increased as peak flow demand increases. As noted in Tables 4 and 5, D_T was the most stable of these trigger variables, and this variable most accurately reflects the response time of the ventilator demand/trigger system.[23,24]

Other factors that may account for the differences observed in trigger-related variables are the setting of trigger sensitivity and the setting of rise time.[25,26] We may have inappropriately set the sensitivity on some ventilators, which could account for their response; however, we do not believe this was the case. With the BEAR 1000 ventilator we had to set the rise time at the zero setting (midpoint on its scale). With a positive rise time setting, airway pressure rapidly exceeded set pressure and repeatedly dropped to baseline during inspiration (Fig. 11).

Table 5. Ventilator Performance Averaged at All Lung Model Peak Flow Rates During Pressure Support at Pressures of 10, 15 and 20 cm H_2O

Ventilator	Pressure Setting (cm H_2O)	P_T (cm H_2O)	D_T (ms)	D_B (ms)	D_{TOT} (ms)	VPF (L/s)	T-PTP (cm $H_2O \cdot s$)	Area I %	Area E (cm $H_2O \cdot s$)	P_E (cm H_2O)	D_E (ms)
BEAR	10	7.32 ± 2.99	104 ± 15	109 ± 29	213 ± 38	1.36 ± 0.20	0.96 ± 0.50	50.86 ± 13.07	5.90 ± 1.40	15.53 ± 4.34	135 ± 16
	15	6.50 ± 2.94	90 ± 13	88 ± 24	178 ± 35	1.46 ± 0.13	0.65 ± 0.43	65.24 ± 8.83*	6.43 ± 1.84	13.23 ± 5.39	128 ± 13
	20	7.12 ± 3.13	84 ± 9*	76 ± 29	160 ± 37	1.48 ± 0.01	0.67 ± 0.41	65.98 ± 6.97	8.42 ± 1.82	14.28 ± 5.32	130 ± 14
Dräger	10	4.63 ± 1.44	62 ± 7	64 ± 28	127 ± 27	1.36 ± 0.17	0.29 ± 0.16	70.03 ± 8.14	5.50 ± 1.28	7.89 ± 2.28	142 ± 36
	15	4.47 ± 1.41	59 ± 8	40 ± 21	99 ± 21*	1.44 ± 0.07	0.20 ± 0.10	76.57 ± 5.35	6.80 ± 1.29	7.90 ± 2.24	111 ± 15
	20	4.45 ± 1.53	64 ± 9	32 ± 12	97 ± 16*	1.49 ± 0.00	0.16 ± 0.07	79.24 ± 4.40*	8.08 ± 1.31*	8.09 ± 2.33	100 ± 14
Galileo	10	5.42 ± 1.57	97 ± 7	62 ± 7	159 ± 11	1.36 ± 0.17	0.43 ± 0.15	67.48 ± 8.10	11.87 ± 0.94	10.15 ± 4.26	316 ± 139
	15	5.28 ± 1.60	94 ± 5	50 ± 7	144 ± 10*	1.50 ± 0.16	0.38 ± 0.14	73.38 ± 5.31	12.70 ± 1.11	9.04 ± 4.41	201 ± 27*
	20	5.32 ± 1.49	93 ± 9	46 ± 9	139 ± 9*	1.63 ± 0.20*	0.35 ± 0.12	75.57 ± 4.79*	13.44 ± 1.35	8.55 ± 4.58	174 ± 9*
PB740	10	4.44 ± 1.10	67 ± 9	30 ± 7	97 ± 7	1.32 ± 0.21	0.18 ± 0.05	75.14 ± 7.03	3.89 ± 1.31	5.86 ± 2.24	117 ± 14
	15	4.37 ± 1.06	66 ± 11	24 ± 5	90 ± 7	1.41 ± 0.11	0.18 ± 0.08	77.29 ± 4.83	4.65 ± 1.36	5.36 ± 2.48	90 ± 9*
	20	4.42 ± 1.02	63 ± 15	24 ± 5	88 ± 11	1.48 ± 0.02	0.17 ± 0.05	78.56 ± 4.70	5.48 ± 1.81	5.36 ± 2.49	80 ± 13*
PB840	10	1.57 ± 0.49	58 ± 4	27 ± 5	84 ± 5	1.41 ± 0.21	0.06 ± 0.02	85.33 ± 1.54	5.45 ± 0.79	4.02 ± 0.55	81 ± 8
	15	1.57 ± 0.30	52 ± 4*	21 ± 3*	73 ± 5*	1.56 ± 0.19	0.06 ± 0.01	86.94 ± 0.73*	6.12 ± 0.66	3.11 ± 0.55*	71 ± 6*
	20	1.33 ± 0.24	51 ± 3*	20 ± 0*	71 ± 3*	1.74 ± 0.17*	0.07 ± 0.09	88.31 ± 0.77*	7.18 ± 0.74*	2.86 ± 0.38*	72 ± 4*
Siemens	10	1.73 ± 0.42	49 ± 3	42 ± 23	91 ± 21	1.24 ± 0.20	0.09 ± 0.06	68.61 ± 10.08	6.19 ± 2.12	8.88 ± 3.65	214 ± 10
	15	1.84 ± 0.50	47 ± 7	28 ± 13	74 ± 13	1.35 ± 0.14	0.06 ± 0.03	74.80 ± 7.30	7.25 ± 2.10	8.49 ± 3.64	141 ± 15
	20	1.85 ± 0.37	49 ± 6	22 ± 7	71 ± 3*	1.40 ± 0.05	0.06 ± 0.02	77.05 ± 5.49	8.68 ± 2.21	8.23 ± 3.63	142 ± 7
TBird	10	4.33 ± 1.69	88 ± 18	117 ± 82	204 ± 93	1.25 ± 0.26	0.36 ± 0.19	64.60 ± 9.60	13.97 ± 3.22	9.64 ± 4.66	483 ± 223
	15	3.85 ± 1.33	73 ± 7	50 ± 18	123 ± 21*	1.35 ± 0.20	0.22 ± 0.09	71.76 ± 7.20	15.25 ± 4.26	8.83 ± 4.33	263 ± 77*
	20	3.94 ± 1.61	77 ± 7	36 ± 10	112 ± 13*	1.42 ± 0.11	0.20 ± 0.09	72.26 ± 9.19	15.65 ± 3.31	7.98 ± 3.74	258 ± 108*

All values are mean ± SD.

*$p < 0.01$ and >10% difference vs 10 cm H_2O pressure support for each ventilator.

P_T = inspiratory trigger pressure. D_T = inspiratory trigger delay time. D_B = inspiratory baseline delay time. D_{TOT} = total inspiratory delay time. VPF = peak flow delivered by the ventilator. T-PTP = trigger pressure-time product. Area I % = area under the airway pressure-time tracing during inspiration expressed as a percentage of the ideal inspiratory area. Area E = area under the airway pressure-time tracing from the beginning of expiration until the return to PEEP. PE = pressure change above peak inspiratory pressure (PIP) during expiration. DE = time delay between the end of inspiration and the return of airway pressure to the plateau pressure. PB = Puritan-Bennett.

Inspiratory Area

This analysis was based on the assumption that the ideal airway pressure curve in pressure targeted ventilation is the immediate increase in pressure from baseline to set level as soon as inspiratory effort begins and sustaining that pressure until inspiration ends.[27] As a result, ventilators with the most rapid delivery of gas flow and the shortest D_{TOT} performed best. Clearly, for some patients this type of inspiratory pressure waveform may not be ideal.[28] However, we did not evaluate the effect of altering inspiratory rise time on the inspiratory area percentage, D_B, D_{TOT}, or T-PTP. We would expect that with a decrease in the rise time an increase in D_B, D_{TOT}, and T-PTP would be observed, resulting in a decrease in the inspiratory area percentage.

Expiratory Variables

The differences observed among ventilators in expiratory variables are a result of a number of issues: the ventilator's ability to sense the end of the lung model's inspiratory phase, the speed of opening the exhalation valve and the resistance to expiratory flow. The supraplateau expiratory pressure (P_E) is most reflective of coordination between the termination of the lung model or patient inspiration and the ending of mechanical inspiration.[29,30] If the lung model ends inspiration after or exactly at the same time the ventilator ends inspiration, there should be a zero P_E if the exhalation valve rapidly opens and the resistance to expiratory flow is not excessive.[31] However, if the lung model ends inspiration before the ventilator, P_E will be greater than zero.[31] The magnitude of the increase depends on the secondary inspiratory termination criteria (pressure increase over set level), the operation of the exhalation valve, and expiratory resistance.[29,31] In all PS evaluations, the lung model terminated inspiration before the ventilator, and as a result all of the above variables affected the P_E. It should be emphasized that this may have been a result of the modified square wave flow pattern of the lung model that did not decrease flow to zero before the end of inspiration. During P A/C, coordination between the ventilator and lung model was better, resulting in a lower P_E

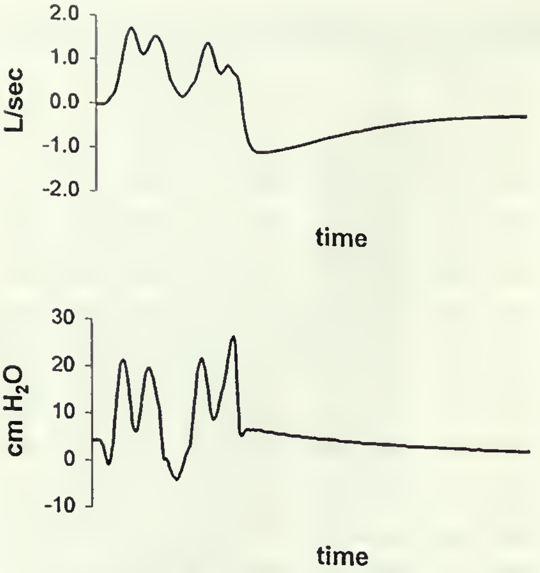

Fig. 11. Flow (top) and airway pressure (bottom) during pressure support (PS) on the BEAR 1000 with the rise time set at maximum, positive end-expiratory pressure at 5 cm H_2O, and inspiratory pressure at 15 cm H_2O.

(see Fig. 5). With all ventilators, increasing lung model peak flow increased P_E. This was a result of greater tidal volume and, as a result, greater elastic recoil of the lung model.

T_E delay (D_E) was most affected by the ventilators' ability to open the exhalation valve and initially decompress the ventilator system. Increasing lung model peak flow and decreasing airway pressure adversely affected D_E on most ventilators, since both of these variables tended to increase end-inspiratory lung model flow.

Expiratory area in any ventilator was most affected by the set pressure or the end-inspiratory lung pressure and lung model peak flow and is a composite reflection of all expiratory variables. It also reflects the overall resistance to exhalation. Area E may be lower with more responsible valves and less expiratory resistance, but these response times and expiratory resistance were not measured. We speculate that those ventilators with the lowest Area E had the most responsive exhalation valves and the least resistance to expiratory flow.

Comparison to Other Studies

Little data comparing the performance of ventilators during PS or P A/C is available.[10,27,32] Hirsch et al[10] found similar trigger pressures at a lung model peak flow of 40 L/min (0.05–3.8 cm H_2O) when a previous generation of ventilators were evaluated at 10 cm H_2O PS with 10 cm H_2O PEEP. However, none of the other variables we evaluated were evaluated by Hirsch. Using a lung model with a 4 L/min peak flow, Nishimura et al[32] identified trigger pressures (1.5 cm H_2O) and total delay times (70 ms), on the Servo 300 set in the pediatric mode, similar to our data. Our data are consistent with that of Bunburaphong et al[27] in their evaluation of bilevel pressure ventilators. They used the same methodology that was used in this evaluation and observed the same variability across ventilators and the same effect of increasing peak lung model flow and set pressure on ventilator performance.

Clinical Implications

The primary clinical impact of this study is the recognition that PS and P A/C are operationally the same except for the inclusion of a backup rate (P A/C) and the method used to terminate inspiration. It is also clear that some of these ventilators can respond well to increased ventilatory demand. That is, these new ventilators can meet patient inspiratory flow demands by increasing delivered flow, while maintaining triggering response time and pressure change (P_{TI}, D_{TOT}) at levels similar to those observed with low inspiratory demand. In addition, when rise time is set at the most rapid setting, these ventilators are able to rapidly pressurize the patient-ventilator system. Of concern are the transitions to exhalation and retarding of the expiratory phase that were observed with many of these ventilators. As shown by Jubran et al[29] and Parthasarathy et al,[30] increases in end-inspiratory pressure above set level are indicative of active exhalation and of increased ventilatory drive. This is a particular problem with PS in ventilators where the flow cycling criteria to exhalation cannot be adjusted. It is also a problem when T_I is set too long during A/C (volume or pressure).

Limitations

The primary limitation of this study is the use of a lung model instead of patients. No lung model can simulate the ventilatory variability observed in patients. As a result, some of the findings of this study may not be directly transferred to patients. Of particular concern is the modified square wave flow pattern of the lung model. Although this flow pattern did exhibit some deceleration, flow did not decrease to zero at end inspiration. Our model may have exaggerated the undesired expiratory variables during PS, since the ventilatory pattern of this lung model is usually only observed in markedly distressed patients. In addition, we did not evaluate variation in inspiratory rise time or inspiratory termination criteria, both critical variables capable of contouring gas delivery during pressure ventilation to the patient's demands. Ventilator performance was also evaluated with only one set of lung model resistance and compliance. As a result, we cannot predict the effect of altered impedence to ventilation but would expect it to be the same for PS and P A/C.

Conclusions

In a given ventilator little difference exists between PS and P A/C. Differences are primarily associated with the transition from inspiration to expiration and during the expiratory phase. Performance differences do exist among the ventilators evaluated. Ventilator performance diminishes at higher lung model peak flows and lower pressure settings.

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Inhaler Technique of Outpatients in the Home

Douglas C Johnson MD and Peggi Robart MEd RRT

OBJECTIVE: Assess the role of evaluation, instruction, and use of spacers by patients using metered dose inhalers (MDIs) in the home. PATIENTS AND SETTING: Patients (age 64 ± 15 y [mean ± standard deviation]) receiving home visits by respiratory care practitioners for oxygen therapy had their inhaler technique checked. INTERVENTIONS AND MEASUREMENTS: A detailed acceptable/unacceptable check-off list was used with 172 patients to evaluate inhaler technique. Patients with poor technique were given instruction and their technique was reassessed. A subgroup of 43 patients was reevaluated on up to 3 visits. RESULTS: Only 18% of patients using MDIs without spacers were rated acceptable with the detailed check list. Instruction improved inhaler technique, but few patients with initially poor technique without spacers developed fully acceptable technique. Improvements made immediately following instruction were lost when patients were reevaluated months later. Few patients received spacers after they were recommended. Technique was markedly better with spacers. Most patients (76%) had initially proper technique with spacers, and most who had poor technique could learn and retain proper technique. CONCLUSIONS: Improper inhaler technique without spacers is very common among patients evaluated at home, and the majority of patients were unable to learn and retain proper technique. Most patients would benefit from using spacers with their inhalers. *Key words: metered dose inhaler, MDI, spacer, technique, chronic obstructive pulmonary disease, COPD.* [Respir Care 2000;45(10):1182–1187]

Background

Inhalation therapy is the primary method of managing asthma and chronic obstructive pulmonary disease (COPD). The metered dose inhaler (MDI) is the most common method of inhalation therapy. Improper inhaler technique is common among asthma and COPD patients,[1] particularly the elderly.[2,3] This can lead to poor delivery of medication to the lung, increased use of inhalers, and poor control of airways obstruction. The frequency of improper inhaler technique in the home setting and the role of home assessment and training of proper inhaler technique are unknown. In an initial survey using a simple acceptable/unacceptable check-off for MDI use, we unexpectedly found that most patients were rated acceptable. This study was undertaken to assess at-home inhaler use using a detailed check list to compare technique with and without spacers and to assess the impact of training on inhaler technique.

Methods

Respiratory therapists (RTs) evaluated MDI technique among patients who were over age 10 and required ongoing visits for home oxygen therapy (supplied by Chartwell Home Therapies, Waltham, Massachusetts). A training session was held with the RTs to demonstrate and review proper inhaler technique and how to use the detailed check list (Fig. 1). Other data collected included patient age and sex, type of spacer, inhaled medications, dosage and frequency of inhaler use, whether a spacer order was needed, and which therapist performed the evaluation. The detailed check list was used with 172 patients (age 64 ± 15 [mean ± standard deviation]), which included 109 females (age 62 ± 17) and 63 males (age 67 ± 12) from May 1995 to February 1996. After each evaluation a progress sheet was sent to the patient's physician indicating the assessment of

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Inhaler Technique Encounter Form (for patients using MDIs)

Name: ________________

Date:__________ Time: __________ Therapist: ______________

MDIs ordered & frequency ______________________________

____

MDI Technique

☐ Acceptable without spacer ☐ Unacceptable without spacer
uses spacer of types* (___, ___)
☐ Acceptable with spacer (___, ___) ☐ Unacceptable with spacer (__, __)
☐ Instruction given. After instruction, technique is
  ☐ Acceptable without spacer ☐ Unacceptable without spacer
  ☐ Acceptable with spacer (__, __) ☐ Unacceptable with spacer (__, __)
☐ Spacer order needed ☐ Ventease order needed

*Aerochamber (A); Aerochamber mask (AM); Azmacort (AZ); Inspirease (I)

If inhaler technique unacceptable, check which step(s) done incorrectly before instruction (first box) and after instruction (second box)

Without spacer
1 ☐ ☐ assemble & remove cap
2 ☐ ☐ shake
3 ☐ ☐ hold upright
4 ☐ ☐ hold about 4 cm away
5 ☐ ☐ wide open mouth
6 ☐ ☐ exhale to FRC or RV
7 ☐ ☐ begin inspiration
8 ☐ ☐ then activate MDI once
9 ☐ ☐ continue to inspire slowly (>3 sec) through mouth to TLC

Aerochamber or Azmacort
10 ☐ ☐ assemble & remove cap
11 ☐ ☐ insert inhaler mouthpiece into end
12 ☐ ☐ shake
13 ☐ ☐ hold upright
14 ☐ ☐ exhale to FRC or RV
15 ☐ ☐ place in mouth and close lips
16 ☐ ☐ activate MDI once
17 ☐ ☐ inspire slowly (so not hear whistle) through mouth to TLC

Inspirease
11 ☐ ☐ insert inhaler nozzle into Inspirease
12 ☐ ☐ shake
10 ☐ ☐ open Inspirease bag
13 ☐ ☐ hold upright
14 ☐ ☐ exhale to FRC or RV
15 ☐ ☐ place Inspirease in mouth and close lips
16 ☐ ☐ activate MDI once (before or after in mouth)
17 ☐ ☐ inspire slowly through mouth to TLC

Last Steps For All:
18 ☐ ☐ hold breath for 5-10 secs
19 ☐ ☐ Wait 20-30 s before next inhalation
20 ☐ ☐ Shake again before next inhalation
21 ☐ ☐ Patient takes correct number of puffs
22 ☐ ☐ Patient reports taking MDIs prescribed number of times daily

Fig. 1. Detailed check list of inhaler technique.

inhaler technique and whether an order for a spacer was needed. To determine the effects of training on retention of proper technique, the first 43 patients who had a follow-up visit at least one month after the initial evaluation were selected for a third evaluation. Forty-three patients (age 68 ± 10) were reevaluated 5.1 ± 1.8 months (mean ± standard deviation) after their initial evaluations. Thirty-seven of these patients were reevaluated 4.0 ± 1.9 months after their second evaluations. Three patients died before the third evaluation and two patients no longer required home oxygen.

Results

There was poor MDI technique among most patients who did not use spacers, but good technique among most who used spacers (Table 1, Fig. 2). Of 126 patients using MDIs without spacers, only 23 (18%) had proper technique. Males and females had similar rates of unacceptable technique: 79% and 83%, respectively. Many patients had a spacer at home but had no idea how to use it or did not know what its purpose was. Common reasons for improper use (see Table 1) included: not holding the MDI about 4

Table 1. Incorrect Procedures among Patients Without and With Spacers*

Procedure		Improper Technique Without Spacers				Improper Technique With Spacers			
		Prior to Instruction		After Instruction		Prior to Instruction		After Instruction	
		No.	(%)	No.	(%)	No.	(%)	No.	(%)
1, 10	Assemble	2	(2)	0	(0)	0	(0)	0	(0)
2, 12	Shake	3	(2)	0	(0)	2	(4)	0	(0)
3, 13	Hold upright	6	(5)	1	(1)	2	(4)	0	(0)
4	About 4 cm away	85	(67)	16	(13)				
5	Open mouth	77	(61)	41	(33)				
6, 14	Exhale to FRC or RV	70	(56)	48	(38)	2	(4)	1	(2)
7	Begin inspiration	11	(9)	7	(6)				
8, 16	Activate MDI once	13	(10)	3	(2)	0	(0)	0	(0)
9, 17	Inspire slowly to TLC	38	(30)	21	(17)	5	(11)	1	(2)
18	Hold breath 5–10 s	58	(46)	27	(21)	6	(13)	1	(2)
19	Wait before next	35	(28)	4	(3)	4	(9)	1	(2)
20	Shake again	1	(1)	0	(0)	0	(0)	0	(0)
21	Correct # of puffs	4	(3)	0	(0)	1	(2)	0	(0)
22	Correct # of times daily	9	(7)	1	(1)	2	(4)	1	(2)

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*Of 126 patients using metered-dose inhalers (MDIs) without spacers, 103 with improper technique before instruction, and 81 after instruction. Of 46 patients using MDIs with spacers, 11 with improper technique before instruction and 1 after instruction. FRC = functional residual capacity. RV = residual volume. TLC = total lung capacity.

cm away from the open mouth, not beginning inhalation from proper volume, not inspiring slowly, and not holding the breath for 5–10 seconds. Following instruction, of the 103 patients with improper technique, most patients still had improper technique. Twenty-four showed proper technique without a spacer and another 9 showed proper technique using a spacer. Forty-seven patients needed orders for spacers.

For 13 of the patients with improper technique, their only problem was not holding the MDI about 4 cm in front of the open mouth. If that step is ignored, 36 of the 126 patients (29%) had proper technique before instruction. Following instruction, of the 90 patients with improper technique, 21 showed proper technique without a spacer and another 4 showed proper technique using the spacer.

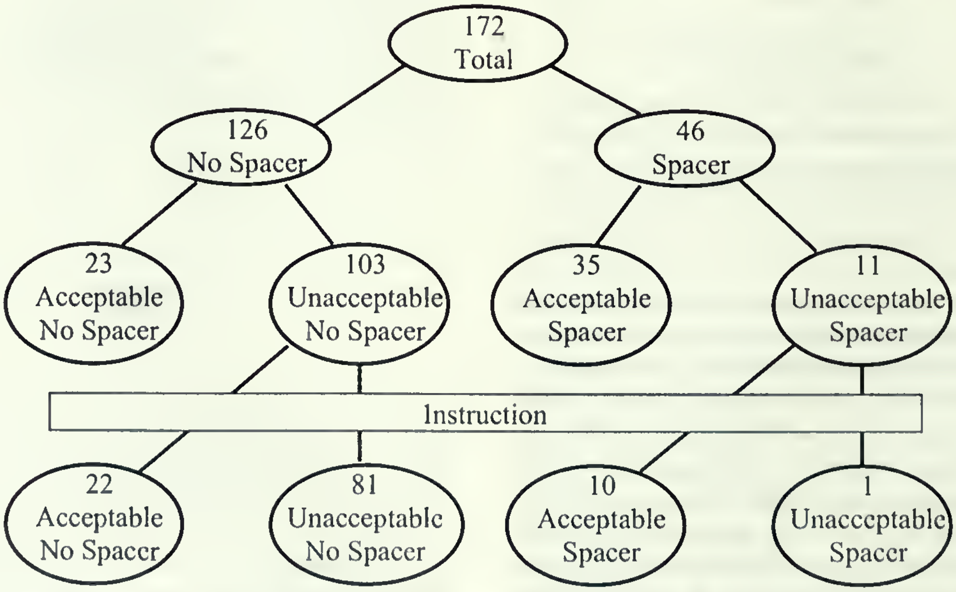

Fig. 2. Results of bronchodilator technique evaluation among 172 outpatients.

Table 2. Incorrect Procedures Before and After Instruction Among Patients Using Metered-Dose Inhalers Without Spacers at First, Second, and Third Evaluations*

		First				Second				Third			
Procedure		Before		After		Before		After		Before		After	
		No.	%	No.	%	No.	%	No.	%	No.	%	No.	%
1	Assemble	0	(0)	0	(0)	0	(0)	0	(0)	0	(0)	0	(0)
2	Shake	2	(6)	0	(0)	1	(3)	0	(0)	0	(0)	0	(0)
3	Hold upright	2	(6)	0	(0)	1	(3)	0	(0)	0	(0)	0	(0)
4	About 4 cm away	30	(91)	7	(21)	30	(91)	6	(18)	20	(77)	6	(23)
5	Open mouth	24	(73)	10	(30)	30	(91)	19	(58)	23	(88)	15	(58)
6	Exhale to FRC or RV	18	(55)	10	(30)	28	(85)	21	(64)	23	(88)	15	(58)
7	Begin inspiration	3	(9)	1	(3)	2	(6)	1	(3)	6	(23)	2	(8)
8	Activate MDI once	5	(15)	2	(6)	3	(9)	1	(3)	3	(12)	0	(0)
9	Inspire slowly to TLC	15	(45)	6	(18)	11	(33)	11	(33)	6	(23)	3	(12)
18	Hold breath 5–10 s	16	(48)	8	(24)	23	(70)	13	(39)	15	(58)	6	(23)
19	Wait before next	10	(30)	3	(9)	18	(55)	6	(18)	10	(38)	1	(4)
20	Shake again	0	(0)	0	(0)	2	(6)	0	(0)	5	(19)	1	(4)
21	Correct # of puffs	2	(6)	0	(0)	0	(0)	0	(0)	1	(4)	0	(0)
22	Correct # of times daily	7	(21)	2	(6)	2	(6)	0	(0)	1	(4)	0	(0)

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*n = 33 for first and second. n = 26 for third. FRC = functional residual capacity. RV = residual volume. TLC = total lung capacity.

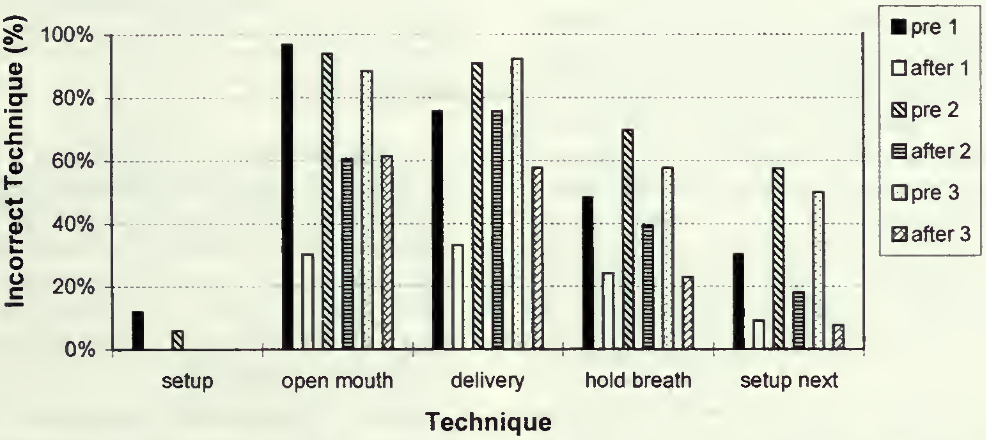

Fig. 3. Incorrect techniques at first, second, and third evaluations among patients using metered-dose inhalers without spacers. Procedures (and steps in detailed check list) include setup (steps 1–3), open mouth (4–5), delivery (6–9), hold breath (18), and setup next (19–20). Percentage of patients: n = 33 for first and second evaluations; n = 26 for third.

Among the 46 patients using spacers, 22 used Aerochamber, 21 used Azmacort, 8 used Inspirease, and 1 used OptiHaler. Six patients used Azmacort as well as another spacer. Of 46 patients using spacers, 11 (24%) had improper technique (see Table 1, Fig. 2). Following instruction, technique was acceptable in 10 of the 11 patients.

Of the 43 patients assessed on multiple visits, 32 had primary diagnoses of COPD and none had primary diagnoses of asthma. Since these patients were selected because they had a second visit (indicating they required oxygen ≥ 6 mo), they might have been sicker than the overall group of patients on home oxygen. While many of these patients improved their technique immediately after instruction, on subsequent visits most patients continued to show the same problems with inhaler use (Table 2, Fig. 3). Of 33 patients not using spacers, 30 had unacceptable technique on both visits 1 and 2. Of 26 patients not using spacers on visit 3, 23 had unacceptable technique. Of the 11 patients using spacers, 4 had unacceptable technique on visit 1, 3 had unacceptable technique on visit 2, and 1 had unacceptable technique on visit 3. With the first visit only 6 patients requested spacers, but only one had received a spacer by the second visit. With the second visit 27 patients requested spacers, but only three had received spacers by the third visit.

Discussion

Few patients using MDIs without spacers have ideal technique. Our finding of only 18% of patients at home having ideal technique is comparable to findings among outpatients,[4] those referred for pulmonary function testing,[1] patients in the emergency room,[5] and hospitalized patients.[6] Improper MDI technique is common, with 60–80% of patients using MDIs incorrectly, depending on the method of assessment, type of inhaler, and patient group.[7]

Proper inhaler technique is important to achieve lung deposition of medication. Using radiotracers, Newman et al[8] found nearly a doubling of lung deposition in patients using correct MDI technique, compared to the patients' usual technique. There is increased bioavailability of inhaled drug by (1) exhaling gently to residual volume rather than to functional residual capacity before inhalation, (2) slow inhalation (compared to fast inhalation), and (3) breath-holding for 10 seconds after inhalation (compared with no breath-holding),[9] and with spacer devices.[10]

There is debate about the proper positioning of MDIs. Increased deposition in the lung occurs when the inhaler is held 2–4 cm from the open mouth, compared to placing the inhaler in the mouth with lips closed.[11,12] However, one study found no difference in efficacy.[13] In our study, most patients (70%) did not position the MDI in front of the open mouth. However, the rate of proper technique only improved from 18% to 28% if both inhaler positions were allowed. Most patients who did not position the MDI in front of the mouth also had other incorrect techniques.

Improper inhaler technique is common among patients of all ages.[1] Our patients were elderly, with 66% over age 60 and 37% over age 70. Poor technique is very common among elderly patients, with a study of 101 elderly patients finding that 45% actuated the MDI after the end of inspiration, and 25% actuated the MDI prior to inhalation.[2] We found that patients frequently had problems with correct position (70%), starting inhalation from FRC or lower (54%), slow inhalation (30%), and breath-hold (45%). Elderly patients with poor hand strength[3] or intellectual impairment[3,14] are more likely to have improper technique.

Knowledge and skills with MDIs are variable among health care providers,[15] with many medical personnel (including physicians, nurses, and RTs) unaware of proper inhaler technique.[16,17] While most RTs had formal instruction on the use of devices at school, only a minority of nurses or physicians had any training. Pharmacists as well often lack formal training and many are unfamiliar with proper technique.[18] A detailed list of proper inhaler technique is essential for patient assessment.

Spacers

We found results that were much different when spacers were used. Seventy-six percent of patients used their MDIs properly with spacers. This improved to 98% following instruction. However, many patients who had spacers did not use them until prompted by the RT. Besides improved ability for correct technique, spacers offer other advantages. The use of spacer devices has been reviewed.[19] The spacer functions as a reservoir in which the aerosol can evaporate and large particles can deposit. Thus, oropharyngeal deposition is reduced and lung deposition improved. Spacer devices improve lung deposition nearly 50% and reduce oropharyngeal deposition by 90%.[8] MDIs given with spacers have been found as effective as nebulized aerosols for the treatment of acute airflow obstruction.[20,21]

Though physicians were sent a form requesting spacers, this proved ineffective. Only 4 of 33 (12%) requests for spacers resulted in patients receiving a spacer. A better solution would be for the RTs to obtain physician approval for the spacer before or at the time of the visit and then to provide the patient with a spacer and instruction. Another reason for poor spacer use may be the lack of reimbursement for the device by most insurance plans. The out-of-pocket expense for most spacers is about $30.

Instruction

Patients are often not provided with proper instruction for inhaler technique. Few pharmacists provide instruction on MDI use.[22] While British Thoracic Society guidelines recommend that all asthmatic patients have their inhaler technique checked prior to discharge from emergency rooms, in one study most emergency room physicians and nurses did not know proper inhaler technique and did not assess patient technique.[23]

Various results have been reported on the effectiveness of training MDI technique. Most studies have looked at the short-term effects of training. Among 125 asthma patients presenting to the emergency department, only 21% performed all steps of MDI use properly.[5] With instruction averaging 8 minutes, nearly all were able to use the MDI properly. In a study of elderly patients without prior MDI use given instruction for MDI use, 62% had correct MDI technique immediately after up to 30 minutes of instruction.[3] Of the correct users, 90% continued to demonstrate correct technique one week later. In a study of 100 stable outpatients with chronic obstructive lung disease there were 38 correct users and 62 incorrect users.[4] Instruction improved MDI technique measured 6–10 weeks later, but the proportion of correct use declined over time.

In the present study most patients without spacers showed some improvement in technique immediately following instruction, but many fewer (22%) learned proper technique for all steps. Even with instruction by a trained professional, only 21% of those with initially improper technique met all criteria for acceptability at the end of a single session. When inhaler technique was reassessed several months later, inhaler technique was as bad as it was prior to instruction. In contrast, instruction was effective for patients using spacers. Following instruction, proper use of inhalers with spacers improved to 98%.

Conclusions

In conclusion, these results show the importance of assessment (using a detailed check list), instruction, and spacers for proper MDI technique. Many patients do not have or use their spacers at home. Most patients not using spacers had improper technique. There was little long-term benefit of instruction to patients who had poor technique without a spacer. In contrast, most patients using spacers had proper technique, and instruction with spacers was very effective. When RTs evaluate patients on home oxygen there is an excellent opportunity to assess and instruct inhaler technique.

ACKNOWLEDGMENTS

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Congenital Cystic Adenomatoid Malformation in the Newborn: Two Case Studies and Review of the Literature

Steven E Sittig RRT and Garth F Asay MD

Congenital cystic adenomatoid malformation (CCAM) is a congenital malformation of the lung that can present on imaging studies as abnormal air, air/fluid-filled cysts, or fluid-filled/solid-appearing cysts. The use of ultrasound in prenatal management has increased the number of cases diagnosed in utero. Early diagnosis is vital in the medical management of CCAM. Outcome varies from hydrops and fetal demise to complete resolution before birth. Many CCAMs diagnosed in utero may decrease in size even if substantial mediastinal shift and lung compression are noted at the time of diagnosis. Once the disorder has been diagnosed, use of serial ultrasound is helpful in providing medical management of the fetus. Two cases of CCAM in the newborn are presented that reflect characteristic clinical features but with distinctly different outcomes, one patient successfully responding to resection and ventilatory support, the other succumbing in the first day of life. The embryology, histology, prenatal and postnatal clinical presentation, and treatment of this malformation are discussed on the basis of a review of the literature. Recent developments in fetal diagnosis and treatment, including fetal surgery, are also presented. We conclude that CCAM should be considered in the differential diagnosis in the presence of respiratory distress and mediastinal shift. It is especially important for respiratory therapists, nurses, and other members of neonatal transport teams to consider CCAM in the differential diagnosis for any patient who presents with respiratory distress and a chest radiograph showing mediastinal shift. The treatment of choice for this lesion is surgical resection by either segmentectomy or lobectomy. Even in cases of relatively asymptomatic patients with CCAM, surgical resection should be considered because of the reported association of carcinoma and unresected CCAM. *Key words: congenital cystic adenomatoid malformation, CCAM, fetal hydrops, pulmonary sequestration, ultrasound, respiratory distress, mediastinal shift, neonate, lobectomy.* [Respir Care 2000;45(10):1188–1195]

Background

Congenital cystic adenomatoid malformation (CCAM) is a rare congenital anomaly representing 25% of all congenital lung malformations and 95% of congenital lung lesions.[1,2] CCAM is thought to occur early in fetal lung development. Absent or aberrant signaling may occur between the bronchioles and the splanchnopleura during the period of bronchial development around the fifth to the eighth weeks of gestation. Although there is no unified agreement on the pathogenesis or the precise embryologic timing, it is agreed that CCAM occurs at the levels of the bronchioles.[3,4] This disorder is characterized by the abnormal development of the terminal respiratory structures and overgrowth of mesenchymal elements. It is this abnormal development that is responsible for the typical adenomatoid or glandlike appearance of the lesion.[1,5]

CCAM was first described in 1949 by Ch'in and Tang.[6] In 1977, Stocker et al[3] proposed classifying CCAM into 3 categories, based on clinical, gross, and microscopic criteria. Recently the disorder was further classified as microcystic or macrocystic in appearance.[7] These defects usually communicate with the bronchial tree and derive blood flow from the pulmonary circulation.[8] CCAM is primarily unilateral but may occur bilaterally. It is now commonly diagnosed in utero by fetal ultrasonography.[9,10]

Initial postnatal presentation may range from asymptomatic (if the cysts are small and unilateral), to mild respi-

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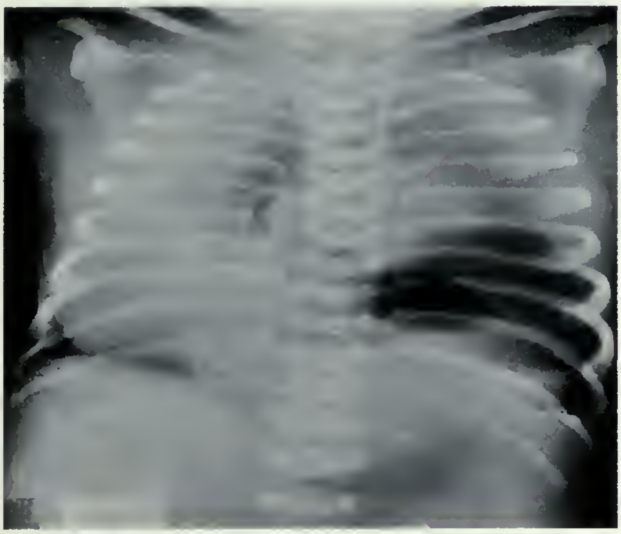

Fig. 1. Case 1. Chest radiograph shows large amount of air in left hemithorax, which may represent a large bulla or a pneumothorax. The mediastinum is shifted to the right.

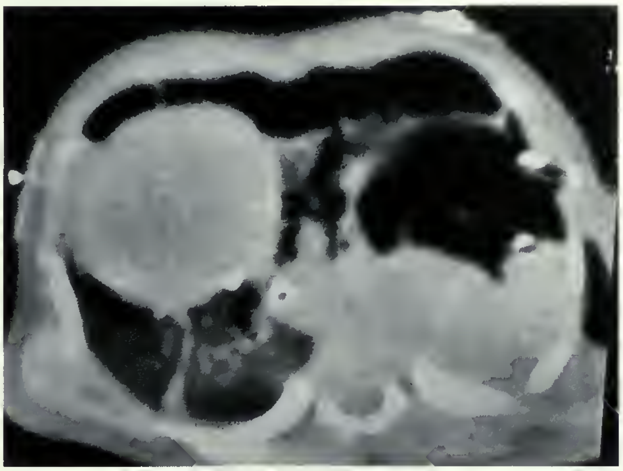

Fig. 2. Case 1. Computed tomography scan shows marked shift of the mediastinum and the heart to the right. Right lung is severely compressed posteriorly. Left lung shows multiple, large, thin-walled, cystlike structures. No vascular markings are seen on the left side. Remainder of left lung is also compressed.

ratory symptoms in childhood, to severe respiratory distress and neonatal death due to pulmonary hypoplasia.[9,11,12]

Case Study Number 1

After a 40-week gestation, a female infant was born by spontaneous vaginal delivery to a 22-year-old woman, gravida 1, para 1, following an uncomplicated pregnancy. Maternal history included limited prenatal care. No fetal ultrasound study was reported.

The patient's initial presentation included a heart rate of 80 beats per minute, a cyanotic appearance, no respiratory effort, and poor muscle tone. Bag-mask ventilation was immediately applied. The infant responded with an increase in heart rate to 120 but remained hypoxic, with an arterial oxygen saturation measured via pulse oximetry (S_{pO_2}) of 30–50%. Physical examination revealed heart sounds shifted to the right, diminished pulses in all extremities, capillary refill of 8 seconds, and dusky overall color. Breath sounds were noted on the right and over the left upper lobe. A chest radiograph (Fig. 1) was taken. The patient was then intubated, and with hand ventilation with a fraction of inspired oxygen (F_{IO_2}) of 1.0 at a rate of 60–100 breaths per minute, there was slight improvement in oxygen saturation, to 50–70%. The postintubation radiograph showed bilateral haziness, with a mediastinal shift to the right.

An umbilical venous catheter was placed and a venous blood gas study revealed a pH of 6.82, partial pressure of carbon dioxide of 100 mm Hg, and partial pressure of oxygen of 36 mm Hg. A needle aspiration of the left chest wall was performed to relieve the mediastinal shift; it produced approximately 40 mL of clear fluid.

A repeat chest radiograph showed a possible pneumothorax on the left and continued mediastinal shift. A left-side chest tube was inserted and clear fluid continued to drain, but an additional chest radiograph was unchanged. Needle aspiration was then performed on the left side, and a large amount of air was removed. The S_{pO_2} increased to 88%, and the heart rate and blood pressure remained stable.

The patient was then prepared for transport and placed on a Biomed MPV 10 (Bio-Med Devices, Guilford, Connecticut), intermittent mandatory ventilation rate 120, peak pressure 25 cm H_2O, positive end-expiratory pressure 5 cm H_2O (25/5), and F_{IO_2} 1.0. A venous blood gas study just before transport showed a pH of 7.1 and a P_{CO_2} of 45 mm Hg. The S_{pO_2} at that time was between 60% and 70%. To facilitate ventilation during transport, the patient was also given pancuronium (Pavulon) and morphine.

On arrival at the neonatal intensive care unit, the patient was placed on a Sensormedics High-Frequency Oscillator (Sensormedics Corporation, Yorba Linda, California) with an initial amplitude of 70, airway pressure 25 cm H_2O, inspiratory time 33%, Hertz 13, and F_{IO_2} 1.0. Three additional chest tubes were placed in the left hemithorax, which initially evacuated air, followed by serosanguineous fluid. The S_{pO_2} briefly increased to above 90%. A repeat chest radiograph again showed persistence of the left-sided collection of air and mediastinal shift. Because it was not possible to adequately evacuate the air from the left side of the chest and relieve the mediastinal shift, the patient was then studied with a computed tomography (CT) scan of the chest and abdomen (Fig. 2), which showed a type I cystic adenomatoid malformation of the left side, with a right mediastinal shift. The right lung was severely compressed.

The patient was returned to the neonatal intensive care unit and high-frequency ventilation was resumed. The chest tube drainage at this time became increasingly bloody, and S_{pO_2} now decreased to the range of 30–50%. A second trial of conventional ventilation resulted in a transient increase in S_{pO_2}, to above 90%.

Since both conventional and high-frequency ventilation had failed, a surgical and extracorporeal membrane oxygenation consult was obtained. However, because of the patient's instability, continued bleeding, and prolonged hypoxia, she was not considered a candidate for either surgery or extracorporeal membrane oxygenation. At 14 hours of age, the parents agreed to withdrawal of life support and the patient died. An autopsy was not obtained, but chest radiograph and CT findings suggested that the fatal lesion was a type I CCAM involving most of the left lung.

Case Study Number 2

After a 37-week gestation, a 3.5 kg female infant was born to a 25-year-old woman, gravida 1, para 1. The pregnancy was complicated by gestational diabetes, which was controlled by diet. A routine ultrasound at 17 weeks of gestation showed no fetal abnormalities.

The child was born by normal spontaneous delivery and had Apgar scores of 4, 6, and 7 at 1, 5, and 10 minutes, respectively. On arrival of the transport team, the patient's S_{pO_2} was 78% on an F_{IO_2} of 1.0. The infant was dusky and desaturated to 60% with stimulation. Breath sounds were diminished and were best heard on the right axilla and left upper lobes. The heart sounds were of a regular rate, without murmur, and heard best on the right. The patient was intubated without difficulty and intravenous access was obtained. A chest radiograph (Fig. 3) showed a marked shift of the heart to the right, with tracheal deviation and opacification of the left lung, sparing the upper lobe. An arterial blood gas study revealed arterial partial pressure of oxygen 40 mm Hg, arterial partial pressure of carbon dioxide of 60 mm Hg, and pH 7.28. During transport the patient was ventilated on a Biomed MPV 10 ventilator, using pressure-control intermittent mandatory ventilation with inspiratory and expiratory pressures of 24 cm H_2O and 4 cm H_2O, respectively, a respiratory rate of 50 breaths per minute, and an inspiratory time of 0.25 second. Venous blood gas analysis indicated a P_{aO_2} of 36 mm Hg, a P_{CO_2} of 36 mm Hg, and a pH of 7.39.

On arrival at the neonatal intensive care unit, a chest and abdominal ultrasound was obtained that suggested a left-sided diaphragmatic hernia. A CT scan showed extensive compression of the lung parenchyma and mediastinal shift due to an intrathoracic solid-appearing mass consistent with a type III CCAM (Fig. 4), and the patient underwent resection of the CCAM. A mass measuring 7.6 cm × 6 cm × 4 cm was removed and confirmed at pathology examination to be a type III CCAM.

The patient remained on mechanical ventilatory support and required only minimal manipulation of the left-sided chest tubes to manage air leaks. She was then weaned from the support system and extubated to room air with no respiratory distress or need for supplemental oxygen.

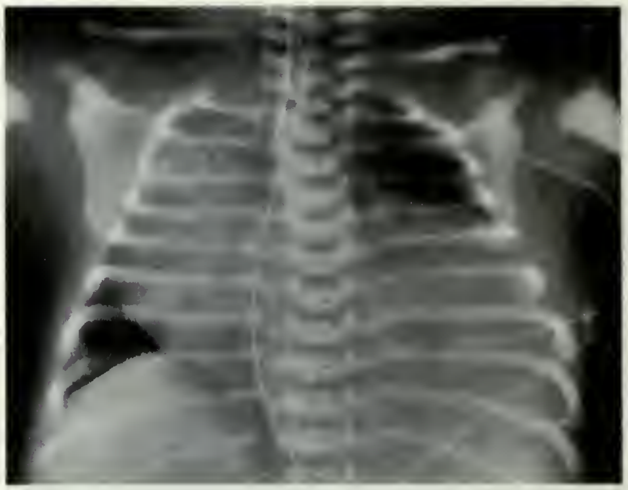

Fig. 3. Case 2. Chest radiograph, with endotracheal tube and feeding tube in place, shows that heart and mediastinum are shifted to the right. There is a soft-tissue density within the left lower lung. Bowel gas pattern is normal.

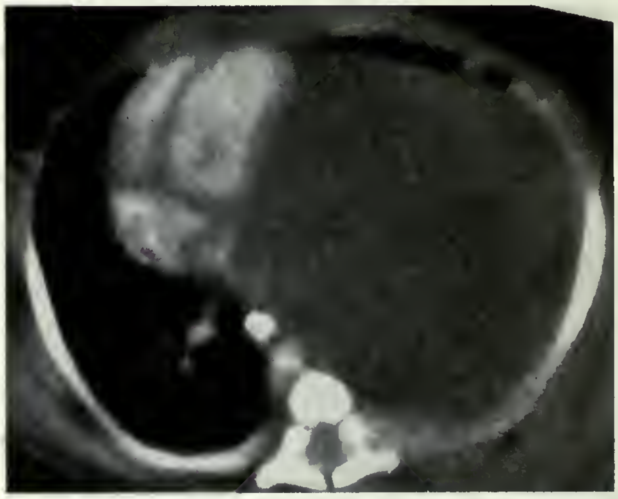

Fig. 4. Case 2. Computed tomography scan shows solid mass in mid-to-lower chest, which lies adjacent to descending thoracic aorta. Mass is causing substantial mediastinal shift to the right, with counterclockwise rotation of the heart.

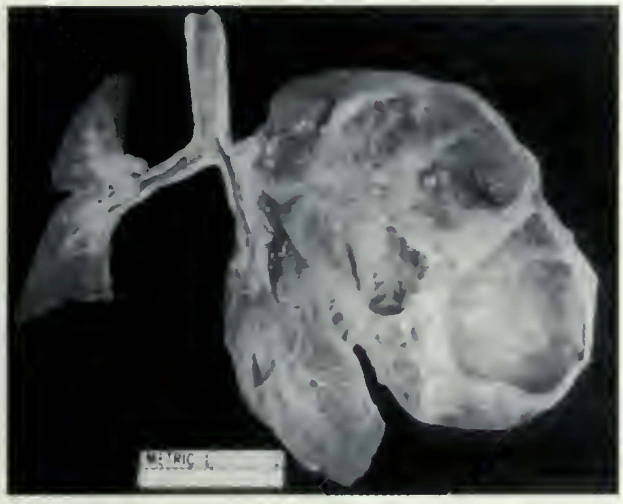

Fig. 5. Type I CCAM of left lung. Note multiple large cystic structures.

Discussion

Histologic Classification

The most commonly seen CCAM is the Stocker type I (Fig. 5), which accounts for approximately 50% of diagnosed cases. This defect is composed of a single cyst or multiple large cysts > 1 cm in diameter, lined by ciliated, pseudostratified columnar epithelium. These cysts commonly produce mediastinal herniation and are usually localized to a single lobe. Normal alveoli may be seen between the cysts.[3]

CCAM Stocker type II, which represents approximately 40% of diagnosed cases, is composed of multiple small cysts of < 1 cm in diameter. These cysts are lined by ciliated cuboidal and columnar epithelium. CCAM type II has also been associated with a higher incidence of other congenital anomalies, such as pulmonary sequestration, congenital heart disease, bilateral renal dysgenesis, and bilateral renal agenesis.[3,6,8]

CCAM Stocker type III represents 10% of diagnosed cases and is characterized by a large space-occupying mass of cysts, each typically 0.3–0.5 cm in diameter, producing a mediastinal shift. Bronchiole-like structures are lined by ciliated cuboidal epithelium and separated by alveolus-sized structures lined by nonciliated cuboidal epithelium.[3] Type III lesions were once considered to be associated with a poor outcome in neonates. Although type III lesions were first associated with poor outcome in neonates, more recent publications indicate that specific aspects of the clinical presentation, such as the presence of fetal hydrops and the degree of pulmonary hypoplasia, are more important factors in the outcome than is the histologic type of malformation.[9,12,13]

Utilization of routine prenatal ultrasound screening has substantially improved early detection and diagnosis of congenital fetal space-occupying lesions such as CCAM.

Prenatal Presentation and Diagnosis

The differential diagnosis for fetal space-occupying lesions includes diaphragmatic hernia, cystic adenomatoid malformations, pulmonary sequestration, bronchogenic cysts, congenital lobar emphysema, and mediastinal cystic teratomas.[14–16] The use of ultrasonography has improved the detection rate of CCAM in utero.[16] The fact that fetal lung tissue exhibits a striking echogenicity easily allows the diagnosis of this and other types of cystic lesions. The majority of CCAM lesions are diagnosed at 16–22 weeks of gestation, presumably when routine ultrasound examinations are performed.[8,10] However, the defect may be so small as to be undetectable by routine ultrasound.

Another peak time of diagnosis occurs around 32 weeks of gestation, probably owing to a policy of routine third-trimester examination or the recognizable development of polyhydramnios.[10] Typically, CCAMs are manifested as hypoechogenic structures in the region of the fetal lung or, in the case of microcystic changes, are associated with hyperechogenic areas. These hyperechogenic areas are caused by small fluid-filled cystic structures and are associated with enhanced dorsal sound intensity. The cystic areas within the mass are so small as to be inapparent sonographically. In these cases, the lesion appears as an echogenic mass and the affected lung is markedly enlarged. Occasionally, macrocystic lesions may be seen on ultrasound examination.[7,8,13]

Large CCAM lesions—those occupying > 50% of the fetal thorax and causing mediastinal shift with cardiovascular compromise, hypoplasia of the uninvolved lung, maternal polyhydramnios, and nonimmune fetal hydrops—are associated with a poor prognosis.[7,8,17] The occurrence of fetal hydrops is thought to be due to the extreme mediastinal shift caused by the CCAM, resulting in vena cava and cardiac compression.[10,17,18] Polyhydramnios is present in approximately half of prenatally diagnosed cases, possibly due to decreased fetal swallowing as a result of esophageal compression by the mass or to decreased absorption of lung fluid by hypoplastic, malformed lungs.[16,19,20]

Differentiating CCAM from the more common congenital diaphragmatic hernia (CDH) can be challenging in a newborn with respiratory distress and an abnormal chest radiograph. The differential diagnosis between CCAM and CDH usually only requires careful ultrasound examination, noting the presence of abdominal organs in the chest cavity or, in the case of a right-sided hernia, possibly noting that a segment of the liver is present past the diaphragm.[15,19]

Distinguishing between CCAM and pulmonary sequestration can be more difficult. Pulmonary sequestration is defined as an immature lung tissue mass that does not communicate with the bronchial tree via an anatomically normal bronchus.[8,14,20,21] Both malformations have a common embryologic derivation resulting from congenital abnormalities of the lung primordia, and they may occur simultaneously.[16,20] Pulmonary sequestration usually appears as a well-circumscribed, uniformly solid-appearing mass in the fetal thorax, similar to CCAM. Pulmonary sequestration is almost always extralobar in the fetus, usually appearing on the left in a posterior and basal location. In these cases, the use of color flow Doppler ultrasonography can be very useful. CCAM is usually supplied by the pulmonary artery, whereas an accessory artery from the aorta supplies pulmonary sequestration.[14,20,21]

Once the clinical diagnosis of CCAM has been made, options in prenatal treatment can be based on gestational age at detection and the clinical status of the fetus.

Prenatal Treatment

The prenatal treatment of CCAM depends on the presentation. The mortality in patients who present with fetal hydrops is up to 68%, as compared with 30% for those without hydrops.[9,13,17,22] For infants diagnosed prenatally in whom hydrops is present, fetal intervention is indicated. In cases of fetal hydrops, thoracoamniotic shunting via a double-lumen pigtail shunt placed under ultrasound guidance may be indicated in cases of large cysts. Serial aspirations of large cysts can also be beneficial, but it is not uncommon for the fluid to reaccumulate; therefore, careful observation is needed, and multiple aspirations may be indicated.[6,13,23] Prevention of compression-related damage to lung tissue increases the chances of successful postpartum surgical intervention; however, in a multicenter study it was shown that shunt operations are associated with a rather high mortality rate (12.5%) and with obstructions and dislocations in 29% of cases.[24] Additional potential risks are premature rupture of the membranes, premature birth, hemorrhage, infection, and injury to the fetus.[25]

In fetuses with less than 32 weeks of gestation who have hydrops and multicystic or predominantly solid microcystic CCAM defects, prenatal resection of the mass may be attempted, but before this decision is made, the potential complication of preterm labor induced by the surgical procedure must be considered.[9,13,17,22] The scientific basis for fetal surgery rests on work done by Harrison et al[26] using fetal lamb models. An intrathoracic balloon was placed into third-trimester lamb fetuses. Inflation of the balloon resulted in pulmonary hypoplasia and death due to pulmonary insufficiency. In contrast, fetal lambs that had a "resection" of the mass by deflation of the balloon in the middle of the third trimester had sufficient lung growth to permit survival at birth.[9]

In 1993 Adzick et al[13] reported on 5 infants with < 32 weeks of gestation who underwent fetal resection for CCAM. Four survived, one of whom was delivered at 26 weeks of gestation. Subsequently, in 1995, Bullard and Harrison[7] reported a series of 6 procedures performed on fetuses ranging from 21 weeks to 30 weeks of gestation. In their series, five survived the operation and were delivered at 26–38 weeks.

The one infant who did not survive postnatally was a 28-week-gestation child who was delivered one week after the intrauterine surgery. The infant succumbed to lung hypoplasia at 40 hours of age.[9]

Fetuses with < 32 weeks of gestation without hydrops should be followed by serial ultrasound examination. It should be kept in mind that 25% of these lesions may regress spontaneously.[11,15,27] In those with more than 32 weeks of gestation without hydrops, early delivery should be considered.[13] Careful routine ultrasound is useful in monitoring the fetal lung defect, and these children should be delivered at tertiary centers where all support services such as obstetricians, neonatologists, and pediatric surgeons are available.[5,7,9] In neither of the case studies presented here was CCAM detected prior to delivery. Although it is rare, caregivers of newborns need to be aware of this disorder and its varied postnatal clinical presentations.

Initial Postnatal Presentation

Approximately 90% of cases of CCAM present in the first year of life. Typically on physical examination there may be prominence of the affected side of the chest, ipsilateral hyperresonance and decreased breath sounds, distant and shifted heart sounds, and hepatosplenomegaly from hyperexpansion of the thorax.[1,28] The most common clinical presentation is some form of progressive respiratory distress, including tachypnea, retractions, grunting, and cyanosis.[1,11,19] If the CCAM communicates with the bronchial tree, progressive expansion of the lesion can result in severe respiratory distress with contralateral shift of the mediastinum, cardiac compression, and compromise of venous return to the heart. This situation may require emergency surgical resection.[29] The diagnosis is generally made from the standard chest radiograph, which shows multiple air-filled, thin-walled cysts of varying size. In severe cases, the lung may be hyperexpanded, with mediastinal shift, flat hemidiaphragm, and herniation of the lung to the contralateral side.

CCAMs may have a solid appearance on chest radiographs if they are of the microcystic type III variety or if there is a type I or type II CCAM with retained fetal lung fluid.[8] The abnormal bronchial connections may cause delayed clearance of fetal lung fluid and a solid-appearing

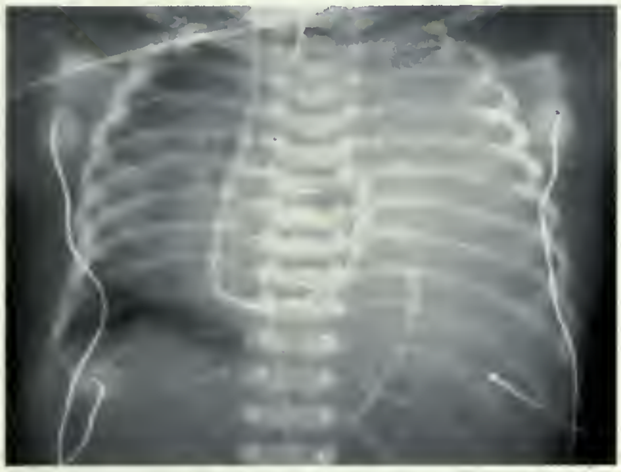

Fig. 6. Chest radiograph of the patient. The left hemithorax is opacified with a midline shift to the right. An endotracheal tube is seen in satisfactory position. The tip of the feeding tube is seen at the level of T-6 and is suspected to be in the stomach, which has herniated into the chest cavity. This is consistent with a Bochdalek type congenital diaphragmatic hernia.

mass initially at birth. Case Study Number 1 demonstrated this feature on the initial chest radiograph. Approximately 30% of fetal lung fluid is removed via the tracheobronchial tree at birth, the remainder being removed via the lymphatic and vascular capillary systems.[30] Therefore, there is radiographic variance in the appearance of this malformation if the defect does not communicate with the bronchial tree or does so only partially. The radiographic appearance would then be of a large pulmonary mass due to a cystic area filled with fluid or of one exhibiting air/fluid levels.[6,8,11] Over the next few hours, as the fetal lung fluid is removed, the CCAM takes on the typical appearance of air-filled cysts.[8,30]

Differentiating CCAM from the more common CDH can be challenging in a newborn with respiratory distress and an abnormal chest radiograph. Use of an orogastric tube can prove helpful in this situation. If the tip of the tube is above the diaphragm (Fig. 6), a diagnosis of CDH can be suspected. If the stomach and intestines are above the diaphragm, CDH is confirmed, whereas if they are below the diaphragm, the diagnosis is probably an intrathoracic process such as CCAM.[5] This feature was found in Case Study Number 2. The different positioning of the orogastric tubes can be noted when comparing Figure 3 and Figure 6.

Mediastinal shift and herniation of the hyperexpanded lung are common findings on the radiograph. In one published report,[11] mediastinal shift was found in 54% of all reported cases and in 55% of surviving infants in this group, which suggests that mediastinal shift alone is a poor predictor of outcome. The use of CT has proved most helpful in determining the actual impact of the defect on the thorax. It is superior to chest radiographs because it can show the amount of mediastinal shift and the anatomic distribution of the defect within the chest.[16,29,30]

CCAMs that do not present immediately after birth with respiratory distress are typically diagnosed later in life as part of a general workup for recurrent pneumonia before the age of 6 years, as an incidental finding on the chest radiograph as part of a fever workup, or for some other reason.[9] In this group of older infants and children the differential diagnosis includes CCAM, lung abscess, and necrotizing pneumonia. The presence of hyperexpansion of the affected lobe and the absence of air bronchograms favor a diagnosis of CCAM. As with CCAM in the newborn period, use of a CT scan with the older patient may be helpful in identifying subtle differences in lung parenchyma resulting from a CCAM that may have resolved in utero.[8,14,27] Once CCAM has been diagnosed, the postnatal treatment plan is primarily dictated by the clinical symptoms of the patient.

Postnatal Treatment

In the symptomatic newborn with CCAM, respiratory distress is often the first feature noted during the child's assessment. Therefore, respiratory stabilization is the first issue to address in preoperative management.[1,11] During mechanical ventilation, frequent assessment of breath sounds along with serial chest radiographs is indicated because the CCAM can become overdistended by positive pressure ventilation. These infants are at risk for the development of air leak syndromes such as pneumothorax, pneumopericardium, and pneumomediastinum.[31,32] For this reason, use of high-frequency ventilation should be considered if the patient is requiring high peak inspiratory pressures and positive end-expiratory pressure levels, and a set ventilator rate.[33,34] The patient should also be closely managed and monitored for any signs of developing persistent pulmonary hypertension of the newborn, the risk of which can be minimized by the avoidance of hypoxia, hypercapnia, and acidosis.[31–35]

The treatment of choice for CCAM is surgical resection. Early surgical removal of the mass is advocated as soon as the patient has been stabilized. Pulmonary resection during infancy is associated with low morbidity and mortality rates. Early resection of CCAM has also been shown to decrease the incidence of complications of recurrent infections and the reported association of carcinomas arising from unresected CCAMs.[36,37]

Lobectomy is preferred to segmental resection. Lobectomy is usually well tolerated, whereas segmental resection has been associated with major complications, including persistent air leaks. The advantage of limited resection is the preservation of normally functioning lung tissue, especially if more than one lobe is involved.[5,6,38] Frenckner and Freyschuss,[33] in a follow-up study of patients after lobectomy for CCAM, found that despite removal of an average of 20% of the total lung volume, the spirometry results were approximately 90% of predicted. This finding would indicate functional compensation by the remaining lung for the resected tissue.[34,38]

Conclusions

Although CCAM is rare, it does represent an important portion of fetal lung lesions. Respiratory therapists and other health professionals who work with neonatal and pediatric patients need to be aware of this malformation. It is especially important for transport team members to be aware of this lesion because of the varied clinical and radiographic appearances this pathology may present. Assessment and early diagnosis and treatment of this disorder are essential. Surgical resection, whether the symptoms are severe or mild, generally results in complete recovery.

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Special Articles

Treating Tobacco Use and Dependence: An Introduction to the US Public Health Service Clinical Practice Guideline

Michael C Fiore MD MPH
for the Guideline Panel and Staff

Overview
Guideline Origins
Style and Structure
Findings and Recommendations
Guideline Update: Advances
Coordination of Care: Institutionalizing the Treatment of Tobacco Dependence
Future Promise

Overview

In America today, tobacco stands out as the agent most responsible for avoidable illness and death. Millions of Americans consume this toxin on a daily basis. Its use brings premature death to almost half a million Americans each year, and it contributes to profound disability and pain in many others. Approximately one third of all tobacco users in this country will die prematurely because of their dependence on tobacco. Unlike so many epidemics in the past, there is a clear, contemporaneous understanding of the cause of this premature death and disability—the use of tobacco.

It is a testament to the power of tobacco addiction that millions of tobacco users have been unable to overcome their dependence and save themselves from its consequences: perpetual worry, unceasing expense, and compromised health. Indeed, it is difficult to identify any other condition that presents such a mix of lethality, prevalence, and neglect, despite effective and readily available interventions.

Despite high, sustained tobacco use prevalence, the response of both clinicians and the United States health care delivery system is disappointing. Studies show that most smokers present at primary care settings, and they are not offered effective assistance in quitting. The smoker's lack of success in quitting and the clinician's reluctance to intervene can be traced to many factors. Until recently, few effective treatments existed, effective treatments had not been identified clearly, and health care systems had not supported their consistent and universal delivery. To single out the clinician for blame would be inappropriate when he or she has typically received neither the training nor support necessary to treat tobacco use successfully.

Current treatments for tobacco dependence offer clinicians their greatest single opportunity to staunch the loss of life, health, and happiness caused by this chronic condition. It is imperative, therefore, that clinicians actively assess and treat tobacco use. In addition, it is imperative that health care administrators, insurers, and purchasers adopt and support policies and practices aimed at reducing tobacco use prevalence. The chief purpose of this document is to provide clinicians, tobacco dependence specialists, health care administrators, insurers, and purchasers, and even tobacco users, with evidence-based recommendations regarding clinical and systems interventions that will increase the likelihood of successful quitting.

Guideline Origins

"Treating Tobacco Use and Dependence," a Public Health Service-sponsored Clinical Practice Guideline, is

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the product of the Tobacco Use and Dependence Guideline Panel ("the panel"), consortium representatives, consultants, and staff. These 30 individuals were charged with the responsibility of identifying effective, experimentally validated tobacco dependence treatments and practices. This guideline updates the 1996 "Smoking Cessation, Clinical Practice Guideline No. 18" that was sponsored by the Agency for Health Care Policy and Research, United States Department of Health and Human Services. The original guideline reflected the extant scientific research literature published between 1975 and 1994.

This guideline was written in response to new, effective clinical treatments for tobacco dependence that have been identified since 1994, and these treatments promise to enhance the rates of successful tobacco cessation. The accelerating pace of tobacco research that prompted the update is reflected by the fact that 3,000 articles on tobacco published between 1975 and 1994 were collected and screened as part of the original guideline. Another 3,000 were published between 1995 and 1999 and contributed to the updated guideline. These 6,000 articles were reviewed to identify a much smaller group of articles that served as the basis for guideline data analyses and panel opinion.

The updated guideline was sponsored by a consortium of 7 federal government and nonprofit organizations:

- Agency for Healthcare Research and Quality
- Centers for Disease Control and Prevention
- National Cancer Institute
- National Heart, Lung, and Blood Institute
- National Institute on Drug Abuse
- Robert Wood Johnson Foundation
- University of Wisconsin Medical School's Center for Tobacco Research and Intervention

All of these organizations have the mission to reduce the human costs of tobacco use. Given the importance of this issue to the health of all Americans, the updated guideline is published by the United States Public Health Service.

Style and Structure

This guideline was written to be relevant to all tobacco users—those using cigarettes as well as other forms of tobacco. Therefore, the terms "tobacco user" and "tobacco dependence" will be used in preference to "smoker" and "cigarette dependence." However, in some cases the evidence for a particular recommendation consists entirely of studies using smokers as subjects. In these instances, the recommendation and evidence refers to "smoking" to communicate the parochial nature of the evidence. In most cases though, guideline recommendations are relevant to all types of tobacco users.

The updated guideline is divided into 8 chapters:

Chapter 1, Overview and Methods: Provides the clinical practice and scientific context of the guideline update project and describes the methodology used to generate the guideline findings.

Chapter 2, Assessment of Tobacco Use: Describes how each patient presenting at a health care setting should have his or her tobacco use status determined and how tobacco users should be assessed for willingness to make a quit attempt.

Chapter 3, Brief Clinical Interventions: Summarizes effective brief interventions that can easily be delivered in a primary care setting. In this chapter, separate interventions are described for the patient who is *willing* to try to quit at this time, for the patient who is not yet willing to try to quit, and for the patient who has recently quit.

Chapter 4, Intensive Clinical Interventions: Outlines a prototype of an intensive tobacco cessation treatment that comprises strategies shown to be effective in this guideline. Because intensive treatments produce the highest success rates they are an important element in tobacco intervention strategies.

Chapter 5, Systems Interventions Relevance to Health Care Administrators, Insurers, and Purchasers: Offers a blueprint to guideline changes in health care coverage and health care administration such that tobacco assessment and intervention become "default options" in health care delivery.

Chapter 6, Evidence: Presents the results of guideline statistical analyses and the recommendations that emanate from them. Guideline analyses address topics such as:

- The efficacy of different pharmacotherapies and counseling strategies.
- The relation between treatment intensities and treatment success.
- Whether screening for tobacco use in the clinic setting enhances tobacco user identification.

The guideline panel made specific recommendations regarding future research on these topics.

Chapter 7, Special Populations: Evaluates evidence on tobacco intervention strategies and efficacy with special populations (eg, women, pregnant smokers, racial and ethnic minorities, hospitalized smokers, smokers with psychiatric comorbidity and chemical dependence, children and adolescents, and older smokers). The guideline panel made specific recommendations for future research on topics relevant to these populations.

Chapter 8, Special Topics: Presents information and recommendations relevant to weight gain after smoking cessation, noncigarette tobacco products, clinician training, economics of tobacco treatment, and harm reduction. The guideline panel formulated specific recommendations regarding future research on these topics.

Findings and Recommendations

The key recommendations of the updated guideline, Treating Tobacco Use and Dependence, based on the literature review and expert panel opinion, follow:

Tobacco dependence is a chronic condition that often requires repeated intervention.

However, effective treatments exist that can produce long-term or even permanent abstinence.

Because effective tobacco dependence treatments are available, every patient who uses tobacco should be offered at least one of these treatments.

- Patients *willing* to try to quit tobacco use should be provided treatments identified as effective in this guideline.
- Patients *unwilling* to try to quit tobacco use should be provided a brief intervention designed to increase their motivation to quit.

It is essential that clinicians and health care delivery systems (including administrators, insurers, and purchasers) institutionalize the consistent identification, documentation, and treatment of every tobacco user seen in a health care setting.

Brief tobacco dependence treatment is effective, and every patient who uses tobacco should be offered at least brief treatment.

There is a strong dose-response relation between the intensity of tobacco dependence counseling and its effectiveness.

Treatments involving person-to-person contact (via individual, group, or proactive telephone counseling) are consistently effective, and their effectiveness increases with treatment intensity (eg, minutes of contact).

Three types of counseling and behavioral therapies were found to be especially effective and should be used with all patients attempting tobacco cessation:

- Provision of practical counseling (problem-solving/skills training).
- Provision of social support as part of treatment (intra-treatment social support).
- Help in securing social support outside of treatment (extra-treatment social support).

Numerous effective pharmacotherapies for smoking cessation now exist. Except in the presence of contraindications, these should be used with all patients attempting to quit smoking.

Five *first-line* pharmacotherapies were identified that reliably increase long-term smoking abstinence rates:

- Bupropion SR.
- Nicotine gum.
- Nicotine inhaler.
- Nicotine nasal spray.
- Nicotine patch.

Two *second-line* pharmacotherapies were identified as efficacious and may be considered by clinicians if first-line pharmacotherapies are not effective:

- Clonidine.
- Nortriptyline.

Over-the-counter nicotine patches are effective relative to placebo, and their use should be encouraged.

Tobacco dependence treatments are both clinically effective and cost-effective relative to other medical and disease prevention interventions.

As such, insurers and purchasers should ensure that:

- All insurance plans include as a reimbursed benefit the counseling and pharmacotherapeutic treatments identified as effective in this guideline.
- Clinicians are reimbursed for providing tobacco dependence treatment just as they are reimbursed for treating other chronic conditions.

Guideline Update: Advances

A comparison of the findings of the year 2000 guideline with the previous 1996 guideline reveals the considerable progress made in tobacco research over the brief period separating these two works. Among many important differences between the two documents, the following deserve special note:

- The updated guideline has produced even stronger evidence of the association between counseling intensity and successful treatment outcomes, and also has revealed evidence of additional efficacious counseling strategies. These include telephone counseling and counseling that helps smokers enlist support outside the treatment context.
- The updated guideline offers the clinician many more efficacious pharmacologic treatment strategies than were identified in the previous guideline. There are now 7 different efficacious smoking cessation medications, allowing the clinician and patient many more treatment options. Further information also is available on the efficacy of combinations of nicotine replacement therapies and pharmacotherapies that are obtained over-the-counter.
- The updated guideline contains strong evidence that smoking cessation treatments shown to be efficacious in this guideline (both pharmacotherapy and counseling) are cost-effective relative to other routinely reimbursed medical interventions (eg, treatment of hyperlipidemia and mammography screening).

The guideline panel concluded, therefore, that smoking cessation treatments should not be withheld from patients when other less cost-effective medical interventions are routinely delivered.

Coordination of Care: Institutionalizing the Treatment of Tobacco Dependence

There is increasing evidence that the success of any tobacco dependence treatment strategy cannot be divorced from the health care system in which it is embedded. Data strongly indicate that the consistent and effective delivery of tobacco interventions requires coordinated interventions. Just as a clinician must intervene with his or her patient, so must the health care administrator, insurer, and purchaser foster and support tobacco dependence treatment as an integral element of health care delivery. Health care purchasers should demand that tobacco intervention be a contractually covered obligation of insurers and providers. Health care administrators and insurers should ensure that clinicians have the training and support and receive the reimbursement necessary to achieve consistent, effective intervention with tobacco users.

Future Promise

About 20 years ago, data indicated that clinicians too frequently failed to intervene with their patients who smoke. Recent data confirm that this picture has not changed markedly over the past two decades. One recent study reported that only 15% of smokers who saw a physician in the past year were offered assistance with quitting, and only 3% were given a follow-up appointment to address this topic. These data are disheartening.

The updated guideline reports a family of findings that creates tremendous tension for change. This guideline reveals that multiple efficacious treatments exist, these treatments can double or triple the likelihood of long-term cessation, many cessation treatments are appropriate for the primary care setting, cessation treatments are more cost-effective than many other reimbursed clinical interventions, and the utilization and impact of cessation treatments can be increased by supportive health system policies (eg, coverage through insurance plans). In sum, the updated guideline identifies and describes scientifically validated treatments and offers clear guidance on how such treatments can be consistently and effectively integrated into health care delivery.

The guideline panel is optimistic that this updated guideline is a harbinger of a new and very promising era in the treatment of tobacco use and dependence. The guideline codifies an evolving culture of health care—one in which every tobacco user has access to effective treatments for tobacco dependence. This new standard of care provides clinicians and health care delivery systems with their greatest opportunity to improve the current and future health of their patients by assisting those addicted to tobacco. Tobacco users and their families deserve no less.

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US Public Health Service Clinical Practice Guideline: Treating Tobacco Use and Dependence

Michael C Fiore MD MPH
for the Guideline Panel and Staff

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  - **Rationale for Initial Guideline Development and Year 2000 Update**
  - **Tobacco Dependence as a Chronic Disease**
  - **Guideline Development Methodology**
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- **5. Systems Interventions. Relevance to Health Care Administrators, Insurers, and Purchasers**
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  - **Recommendations Regarding Specific Pharmacotherapies: First-Line Medications**
  - **Recommendations Regarding Specific Pharmacotherapies: Second-Line Medications**
  - **Pharmacotherapies Not Recommended by the Guideline Panel**
  - **Pharmacotherapy for Treating Tobacco Dependence: Issues Relevant to Use**
  - **Recommending Specific Pharmacotherapy for Specific Patient Subgroups**
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  - **Racial and Ethnic Minorities**

Hospitalized Smokers
Smokers With Psychiatric Comorbidity and/or Chemical Dependence
Children and Adolescents
Older Smokers
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Weight Gain After Smoking Cessation
Noncigarette Tobacco Products
Clinician Training
Economic Aspects of Tobacco Dependence Treatments and Health Systems Interventions
Alternative Treatment Goals: Harm Reduction
Guideline Panel
Guideline Staff

Abstract

Treating Tobacco Use and Dependence, a Public Health Service-sponsored Clinical Practice Guideline, is a product of the Tobacco Use and Dependence Guideline Panel ("the panel"), consortium representatives, consultants, and staff. These 30 individuals were charged with the responsibility of identifying effective, experimentally validated tobacco dependence treatments and practices. The updated guideline was sponsored by a consortium of seven Federal Government and nonprofit organizations: the Agency for Healthcare Research and Quality (AHRQ), Centers for Disease Control and Prevention (CDC), National Cancer Institute (NCI), National Heart, Lung, and Blood Institute, National Institute on Drug Abuse, Robert Wood Johnson Foundation, and University of Wisconsin Medical School's Center for Tobacco Research and Intervention. This guideline is an updated version of the 1996 *Smoking Cessation Clinical Practice Guideline No. 18* that was sponsored by the Agency for Health Care Policy and Research (now the AHRQ), United States Department of Health and Human Services. The original guideline reflected the extant scientific research literature published between 1975 and 1994.

The updated guideline was written because new, effective clinical treatments for tobacco dependence have been identified since 1994. The accelerating pace of tobacco research that prompted the update is reflected in the fact that 3,000 articles on tobacco were identified as published between 1975 and 1994, contributing to the original guideline. Another 3,000 were published between 1995 and 1999 and contributed to the updated guideline. These 6,000 articles were screened and reviewed to identify a much smaller group of articles that served as the basis for guideline data analyses and panel opinion.

This guideline contains strategies and recommendations designed to assist clinicians, tobacco dependence treatment specialists, and health care administrators, insurers, and purchasers in delivering and supporting effective treatments for tobacco use and dependence. The recommendations were made as a result of a systematic review and analysis of the extant scientific literature, using meta-analysis as the primary analytic technique. The strength of evidence that served as the basis for each recommendation is clearly indicated in the guideline. A draft of the guideline was peer-reviewed prior to publication, and the comments of 70 external reviewers were incorporated into the final document. The key recommendations of the updated guideline, *Treating Tobacco Use and Dependence,* based on the literature review and expert panel opinion, are as follows:

1. Tobacco dependence is a chronic condition that often requires repeated intervention. However, effective treatments exist that can produce long-term or even permanent abstinence.
2. Because effective tobacco dependence treatments are available, every patient who uses tobacco should be offered at least one of these treatments:
   - Patients *willing* to try to quit tobacco use should be provided with treatments identified as effective in this guideline.
   - Patients *unwilling* to try to quit tobacco use should be provided with a brief intervention designed to increase their motivation to quit.

3. It is essential that clinicians and health care delivery systems (including administrators, insurers, and purchasers) institutionalize the consistent identification, documentation, and treatment of every tobacco user seen in a health care setting.
4. Brief tobacco dependence treatment is effective, and every patient who uses tobacco should be offered at least brief treatment.
5. There is a strong dose-response relation between the intensity of tobacco dependence counseling and its effectiveness. Treatments involving person-to-person contact (via individual, group, or proactive telephone counseling) are consistently effective, and their effectiveness increases with treatment intensity (eg, minutes of contact).
6. Three types of counseling and behavioral therapies were found to be especially effective and should be used with all patients attempting tobacco cessation:
    - Provision of practical counseling (problem-solving/skills training);
    - Provision of social support as part of treatment (intra-treatment social support); and
    - Help in securing social support outside of treatment (extra-treatment social support).
7. Numerous effective pharmacotherapies for smoking cessation now exist. Except in the presence of contraindications, these should be used with all patients attempting to quit smoking.
    - Five *first-line* pharmacotherapies were identified that reliably increase long-term smoking abstinence rates:
        - –Bupropion SR
        - –Nicotine gum
        - –Nicotine inhaler
        - –Nicotine nasal spray
        - –Nicotine patch
    - Two *second-line* pharmacotherapies were identified as efficacious and may be considered by clinicians if first-line pharmacotherapies are not effective:
        - –Clonidine
        - –Nortriptyline
    - Over-the-counter nicotine patches are effective relative to placebo, and their use should be encouraged.
8. Tobacco dependence treatments are both clinically effective and cost-effective relative to other medical and disease prevention interventions. As such, insurers and purchasers should ensure that:
    - All insurance plans include as a reimbursed benefit the counseling and pharmacotherapeutic treatments identified as effective in this guideline, and
    - Clinicians are reimbursed for providing tobacco dependence treatment just as they are reimbursed for treating other chronic conditions.

The updated guideline is divided into eight chapters that provide an overview including methods (Chapter 1), information on the assessment of tobacco use (Chapter 2), brief clinical interventions, both for patients willing and unwilling to make a quit attempt at this time (Chapter 3), intensive clinical interventions (Chapter 4), systems interventions for health care administrators, insurers, and purchasers (Chapter 5), the scientific evidence supporting the guideline recommendations (Chapter 6), and special populations and topics (Chapters 7 and 8).

A comparison of the findings of the updated guideline with the original guideline reveals the considerable progress made in tobacco research over the brief period separating these two publications. Tobacco dependence is now increasingly recognized as a chronic disease, one that typically requires ongoing assessment and repeated intervention. In addition, the updated guideline offers the clinician many more efficacious treatment strategies than were identified in the original guideline. There are now seven different efficacious agents in the smoking cessation pharmacopoeia, allowing the clinician and patient many different medication options. In addition, recent evidence has identified new, efficacious counseling strategies. In particular, proactive telephone counseling is efficacious, as is counseling that helps smokers attain social support outside of the treatment context. The updated guideline also reveals greater evidence of the strong dose-response relation between counseling intensity and the likelihood of long-term abstinence from tobacco. Indeed, the data are compelling that pharmacologic and counseling treatment each independently boost cessation success; these data suggest that optimal cessation outcomes may require the combined use of both counseling and pharmacotherapy.

Finally, there is increasing evidence that the success of any tobacco dependence treatment strategy or

effort cannot be divorced from the health care system in which it is embedded. Data strongly indicate that effective tobacco interventions require *coordinated interventions*. Just as the clinician must intervene with his or her patient, so must the health care administrator, insurer, and purchaser foster and support tobacco intervention as an integral element of health care delivery. Health care administrators and insurers should ensure that clinicians have the training and support, and receive the reimbursement necessary to achieve consistent, effective intervention with tobacco users.

One important conclusion of this guideline is that the most effective way to move clinicians to intervene is to provide them with information regarding multiple efficacious treatment options and to ensure that they have ample institutional support to use these options. Indeed, in this guideline, the panel encourages a culture of health care in which failure to treat tobacco use—the chief cause of preventable disease and death—constitutes an inappropriate standard of care. *Key words: tobacco, dependence, addiction, nicotine, cessation, Bupropion, Nortriptyline, patch.* [Respir Care 2000;45(10):1200–1262]

1. Overview and Methods

Introduction

Tobacco use has been cited as the chief avoidable cause of illness and death in our society, responsible for more than 430,000 deaths in the United States each year.[1] Smoking is a known cause of cancer, heart disease, stroke, complications of pregnancy, and chronic obstructive pulmonary disease (COPD).[2] Given the health dangers it presents and the public's awareness of those dangers, tobacco use remains surprisingly prevalent. Recent estimates are that 25% of adult Americans smoke.[3–5] Moreover, smoking prevalence among adolescents has risen dramatically since 1990,[6] with more than 3,000 additional children and adolescents becoming regular users of tobacco each day.[7,8] As a result, a new generation of Americans has become dependent upon tobacco and is at risk for the extraordinarily harmful consequences of tobacco use.

Tobacco use is not only dangerous to individuals, it also results in staggering societal costs. The estimated smoking-attributable cost for medical care in 1993 was more than $50 billion,[9] and the cost of lost productivity and forfeited earnings due to smoking-related disability was estimated at $47 billion per year.[10]

Despite the tragic health consequences of using tobacco, clinicians often fail to assess and treat tobacco use consistently and effectively. For instance, in 1995 smoking status was identified in only about 67% of clinic visits, and smoking cessation counseling was provided in only 21% of smokers' clinic visits.[11] Moreover, treatment is typically offered only to patients already suffering from tobacco-related diseases.[11] This failure to assess and intervene consistently with tobacco users exists in the face of substantial evidence that even brief smoking cessation treatments can be effective.[12–15]

This guideline concludes that tobacco use presents a rare confluence of circumstances: (1) a highly significant health threat, (2) a disinclination among clinicians to intervene consistently, and (3) the presence of effective interventions. This last point is buttressed by evidence that smoking cessation interventions, if delivered in a timely and effective manner, significantly reduce the smoker's risk of suffering from smoking-related disease.[16,17] Indeed, it is difficult to identify any other condition that presents such a mix of lethality, prevalence, and neglect, despite effective and readily available interventions.

Finally, significant barriers exist that interfere with clinicians' assessment and treatment of smokers. Many clinicians lack knowledge about how to identify smokers quickly and easily, which treatments are efficacious, how such treatments can be delivered, and the relative efficacies of different treatments.[18] Additionally, they may fail to intervene because of inadequate clinic or institutional support for routine assessment and treatment of tobacco use[11,19,20] and for other reasons such as time constraints.[21,22]

Rationale for Initial Guideline Development and Year 2000 Update

In the early 1990s, the Agency for Health Care Research and Policy (now the Agency for Healthcare Research and

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Parts of the original document are not reprinted herein but are available at http://www.osophs.dhhs.gov/tobacco. The omitted sections are: Appendix A: Helpful Web site addresses. Appendix B: Coding information regarding the diagnosis of and billing for tobacco dependence treatment. Appendix C: Financial disclosures for panel members, consultants, and senior project staff. Glossary. Index.

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Quality [AHRQ]) convened an expert panel to develop the *Smoking Cessation Clinical Practice Guideline No. 18* (the "guideline") in the AHCPR series of Clinical Practice Guidelines. The need for this guideline was based on several factors, including tobacco use prevalence, related morbidity and mortality, the economic burden imposed by tobacco use, variation in clinical practice, availability of methods for improvement of care, and availability of data upon which to base recommendations for care.

Since the guideline was published in 1996, it has become a popular document. More than 1 million copies of the guideline and its affiliated products have been disseminated. Guideline recommendations have inspired changes in diverse health care settings such as health maintenance organizations and Veteran's Administration hospitals. The original guideline continues to provide a framework for educating clinicians, administrators, and policy makers about the importance of tobacco dependence and its treatment. It has stimulated discussions that address the development of tobacco dependence treatment programs at the federal and state levels and by professional medical organizations.

Significant new research on tobacco use and its treatment has appeared since the publication of the original guideline. As a result of this new research, as well as an increasing recognition that tobacco interventions must become an integral part of health care delivery, the expert panel that developed the 1996 *Smoking Cessation Clinical Practice Guideline No. 18* was reconvened in 1998 to conduct an update. This guideline update is sponsored by a consortium of private and public partners, including the AHRQ, National Cancer Institute (NCI), National Heart, Lung, & Blood Institute, National Institute on Drug Abuse, Office on Smoking and Health at the Centers for Disease Control and Prevention (CDC), Robert Wood Johnson Foundation, and University of Wisconsin Medical School's Center for Tobacco Research and Intervention.

The original guideline addressed barriers to effective smoking cessation intervention on the basis of a careful evaluation and synthesis of relevant scientific evidence. The guideline comprised specific evidence-based recommendations to guide clinicians and smoking cessation specialists in their tobacco intervention efforts. Additional specific recommendations guided insurers, purchasers, and health care administrators in their efforts to develop and implement institutional and policy changes that support the reliable assessment and treatment of tobacco dependence.

The updated guideline, *Treating Tobacco Use and Dependence,* a Public Health Service-sponsored Clinical Practice Guideline, provides recommendations based on evidence published through January 1, 1999. This new title underscores three truths about tobacco use.[23] First, all tobacco products, not just cigarettes, exact devastating costs on the Nation's health and welfare. Second, for most users, tobacco use results in true drug dependence, one comparable to the dependence caused by opiates, amphetamines, and cocaine.[24] Third, chronic tobacco use warrants clinical intervention just as do other addictive disorders.[18,25]

Most tobacco users in the United States are cigarette smokers. As a result, the majority of clinician attention and research in the field has focused on the treatment and assessment of smoking. However, clinicians should intervene with all tobacco users, not just with smokers. To foster a broad implementation of this guideline, every effort has been made to describe interventions so that they are relevant to all forms of tobacco use. In some sections of this guideline (eg, sections of Chapter 6), the term "smoker" is used instead of "tobacco user." The use of the term "smoker" merely means that all relevant evidence for a recommendation arises exclusively from studies of cigarette smokers. Additional discussion of noncigarette forms of tobacco use is found in Chapter 8.

It is important to note that other guidelines and analyses on the treatment of tobacco dependence have been published, including those from the American Psychiatric Association,[26] the American Medical Association,[27] the United Kingdom Guideline,[28] and those published by the Cochrane Collaboration.[29]

Tobacco Dependence as a Chronic Disease

Tobacco dependence shows many features of a chronic disease. Although a minority of tobacco users achieves permanent abstinence in an initial quit attempt, the majority persist in tobacco use for many years and typically cycle through multiple periods of relapse and remission. A failure to appreciate the chronic nature of tobacco dependence may undercut clinicians' motivation to treat tobacco use consistently.

Epidemiologic data suggest that more than 70% of the 50 million smokers in the United States today have made at least one prior quit attempt, and approximately 46% try to quit each year.[4] Unfortunately, most of these efforts are unsuccessful; among the 17 million adults who attempted cessation in 1991, only about 7% were still abstinent 1 year later.[30,31] These discouraging statistics have led many clinicians to report that they feel ineffective in the treatment of tobacco dependence.

Moreover, as described in a recent editorial,[32] much smoking cessation research and clinical practice over the last three decades has focused on identifying the ideal intervention that would turn all smokers into permanent nonsmokers. This effort may have inadvertently communicated two messages of dubious validity: first, that there is one treatment that will be effective for virtually all smokers, and, second, that success should be defined only on the basis of permanent abstinence. These messages may have masked the true nature of tobacco addiction; it is typically a chronic disorder that carries with it a vulnerability to relapse that persists for weeks, months, and perhaps years.

A more productive approach is to recognize the chronicity of tobacco dependence. A chronic disease model has many appealing aspects. It recognizes the long-term nature of the disorder with an expectation that patients may have periods of relapse and remission. If tobacco dependence is recognized as a chronic condition, clinicians will better understand the relapsing nature of the ailment and the requirement for ongoing, rather than just acute, care. Clinicians also should recognize that despite the potential for relapse, numerous effective treatments are now available and described in this guideline.

A chronic disease model emphasizes for clinicians the importance of counseling and advice. Although most clinicians are comfortable in counseling their patients about diabetes, hypertension, or hyperlipidemia, many believe that they are ineffective in providing counseling to patients who use tobacco. As with these chronic disorders, clinicians encountering a patient dependent on tobacco must be encouraged to provide that patient with simple counseling advice, support, and appropriate pharmacotherapy. In updating the guideline, the panel has presented evidence-based analytic findings in a format accessible and familiar to practicing clinicians. Although this should aid clinicians in the assessment and treatment of tobacco users, clinicians should remain cognizant that relapse is likely and that it reflects the chronic nature of dependence, not their personal failure, nor a failure of their patients.

Guideline Development Methodology

Introduction. Panel recommendations are intended to provide clinicians with effective strategies for treating patients who use tobacco. Recommendations were influenced by two goals. The first was to identify clearly efficacious treatment strategies. The second was to formulate and present recommendations so that they can be implemented easily across diverse clinical settings and patient populations.

The guideline is based on two systematic reviews of the available scientific literature. The first review occurred during the creation of the original guideline published in 1996 and included literature published from 1975 through 1994. The second review was conducted for the updated guideline and included literature from 1995 through January 1, 1999. The two reviews were then combined into a single database.

The panel identified randomized placebo/comparison controlled trials as the strongest level of evidence for evaluation of treatment efficacy. Thus, evidence derived from randomized controlled trials serves as the basis for meta-analyses and for almost all recommendations contained in this guideline. However, the panel occasionally made recommendations in the absence of randomized controlled trials. It did so when faced with an important clinical practice issue for which persuasive evidence existed. When the panel considered evidence other than randomized controlled trials, it did not restrict itself to articles that otherwise met the inclusion criteria. For example, for recommendations that were not based on meta-analyses, the panel reviewed some articles published after January 1, 1999. This guideline clearly identifies the level or strength of evidence that serves as the basis for each of its recommendations.

Topics Included in the Guideline. The panel identified tobacco use as the targeted behavior and tobacco users as the clinical population of interest. Tobacco dependence treatments were evaluated for efficacy as were interventions aimed at modifying both clinician and health care delivery system behavior.

Interventions for the primary prevention of tobacco use were not examined in detail, with the exception of interventions directly relevant to clinical practice. Because of the importance and complexity of the primary prevention of tobacco initiation, the panel recommends that primary prevention be addressed in a separate clinical practice guideline. Readers may also refer to the 1994 Surgeon General's Report, *Preventing Tobacco Use Among Young People*.[63] In addition, community-level interventions (eg, mass media campaigns) that are not usually implemented in primary care practice settings are not addressed. For more information on community-based tobacco use prevention, refer to the *Centers for Disease Control and Prevention Guide to Community Preventive Services* (available in 2000).

This guideline is designed for three main audiences: primary care clinicians, tobacco dependence treatment specialists, and health care administrators, insurers, and purchasers. Additionally, the guideline is designed to be used in a wide variety of clinical practice settings, including private practices, academic health centers, managed care settings and health maintenance organizations, public health department clinics, hospitals, and school or work site clinics.

At the start of the update process, guideline panel members, outside experts, and consortium representatives were consulted to determine those aspects of the original guideline that required updating. These consultations resulted in the following chief recommendations that guided the update efforts: (1) to update any recommendations from the original guideline likely to be affected by new research findings, (2) to provide information and recommendations on health systems changes relevant to tobacco cessation including the cost-effectiveness of tobacco cessation, (3) to summarize the literature and make recommendations regarding special populations, and (4) to address content

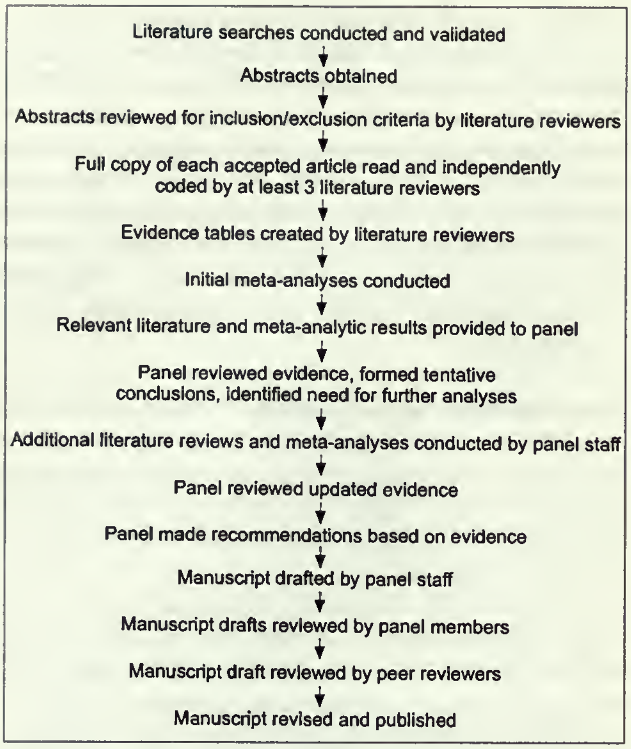

Literature searches conducted and validated
Abstracts obtained
Abstracts reviewed for inclusion/exclusion criteria by literature reviewers
Full copy of each accepted article read and independently coded by at least 3 literature reviewers
Evidence tables created by literature reviewers
Initial meta-analyses conducted
Relevant literature and meta-analytic results provided to panel
Panel reviewed evidence, formed tentative conclusions, identified need for further analyses
Additional literature reviews and meta-analyses conducted by panel staff
Panel reviewed updated evidence
Panel made recommendations based on evidence
Manuscript drafted by panel staff
Manuscript drafts reviewed by panel members
Manuscript draft reviewed by peer reviewers
Manuscript revised and published

Fig. 1. Guideline development process. These steps were taken in drafting both the original and updated guidelines.

areas and models of treatment for which little data existed during the development of the original guideline.

Guideline Development Process. The original guideline development process was initiated in late 1993. This update was initiated in mid-1998. The methodology was consistent between the two efforts except where specifically identified below (Fig. 1).

Selection of Evidence. Published, peer-reviewed, randomized controlled studies were considered to constitute the strongest level of evidence in support of guideline recommendations. This decision was based on the judgment that randomized controlled trials provide the clearest, scientifically sound basis for judging comparative efficacy. The panel made this decision recognizing the limitations of randomized controlled trials, particularly considerations of generalizability with respect to patient selection and treatment quality.

Literature Review and Inclusion Criteria. Approximately 6,000 articles were reviewed to identify evaluable literature—3,000 during the original project and another 3,000 during the update. These articles were obtained through searches of electronic databases and reviews of published abstracts and bibliographies. An article was deemed appropriate for meta-analysis if it met the criteria for inclusion established *a priori* by the panel. These criteria were that the article: (A) reported the results of a randomized, placebo/comparison controlled trial of a tobacco-use treatment intervention randomized on the patient level, (B) provided follow-up results at a time point at least 5 months after the quit date, (C) was published in a peer-reviewed journal, (D) was published between January 1, 1975 and January 1, 1999, and (E) was published in English. Additionally, articles screened during the update were screened for relevance to economic or health systems issues. As a result of the original and updated literature reviews, more than 180 articles were identified for possible inclusion in a meta-analysis, and more than 500 additional articles were examined by the panel. These articles were used in the consideration and formulation of panel recommendations that were not supported by meta-analyses. The literature search for the update project was validated by comparing the results against a search conducted by the CDC, by a review of the database by the expert panel, and by requesting articles pertaining to special topics from experts in the field.

It is important to note that because of a faithful application of article screening criteria in the updated guideline, some of the studies that were included in the original guideline were not included in the updated analyses. This resulted in an inability to perform certain analyses that had previously been conducted (eg, analysis of the different types of self-help).

When individual authors published multiple articles meeting the meta-analytic inclusion criteria, the articles were screened to determine whether they contained unique data. Where two articles reported data from the same group of subjects, both articles were used to elicit the complete trial data for the analyses.

Preparation of Evidence Tables. Three reviewers independently read and coded each article that met inclusion criteria. The reviewers coded the treatment characteristics that were used in data analyses (see Table 6 in Chapter 6). The same general coding procedure employed during the original guideline process was employed during the update. Where adjustments to the coding process were made, articles from the original process were re-coded to reflect the changed coding (eg, more refined counseling and behavioral therapy designations were used during the update to capture more specific counseling practices.) The reviewers then met and compared coding. Any discrepancies that could not be resolved were adjudicated by the project director, panel chair, and/or senior scientific consultant. The data were then compiled and used in relevant analyses. As a test of the coding process for the update, interrater reliability analyses were conducted on four coded intervention categories: type of format, type of clinician, type of counseling and behavioral therapies, and level of person-to-person contact. Using the proportional overlap method[33] for format, clinician and counseling and behavioral therapies, and the nominal response method[34] for level of person-to-person contact, reliability analyses were conducted on 16 studies that had all been coded by the same three reviewers. Coded data were sampled from the preadjudicated ratings made by the three reviewers who coded the greatest number of articles. Studies and intervention categories were selected after coding, so reviewers were unaware of the data to be analyzed for reliability. Results revealed the following chance-corrected inter-rater reliabilities for each of the tested categories: $\kappa = 0.73$ for format, $\kappa = 0.72$ for clinician, $\kappa = 0.77$ for counseling and behavioral therapies, and $\kappa = 0.78$ for level of person-to-person contact.

Outcome Data. To meet inclusion criteria for the meta-analyses, a study was required to provide outcome data with follow-up at least 5 months after the designated quit day. Five months was chosen to balance the needs for (A) a large pool of studies for meta-analyses and (B) the desire to examine only clinically important outcomes (ie, long-term abstinence). When quit rates were provided for multiple long-term end points, efficacy data from the end point closest to 6 months were used, so long as they did not exceed 3 years. Virtually all cessation analyses in this guideline were done on these long-term outcome data. (One exception is that the meta-analysis of cessation treatments in pregnant women allowed somewhat shorter follow-up periods because of the desire for preparturition data.)

Panel staff also coded biochemical confirmation of self-reported abstinence. Previous guideline analyses show that studies with and without biochemical confirmation yield similar meta-analysis results. Therefore, meta-analyses presented in the guideline reflect a pooling of these studies. The only exception to this was in the pregnancy analyses. Data suggest that self-reported abstinence rates may be less reliable in pregnant women; therefore, the pregnancy meta-analysis included only abstinence data that were biochemically confirmed.[35–37]

Two types of follow-up data were included in the analyses to index treatment efficacy. The first and preferred type of data were intent-to-treat data, in which the denominator was the number of patients randomized to treatment and the numerator was the number of abstinent patients contacted at follow-up. The second type of acceptable data were similar, except that the denominator consisted only of patients who had completed treatment. Other types of follow-up data were not included in analyses (eg, studies in which the denominator included only those subjects contacted during follow-up).

Studies were coded for how the outcome measures were reported, "point prevalence," "continuous," or "unknown." If abstinence data were based on smoking occurrence within a set time period (usually 7 d) prior to a follow-up assessment, the outcome measure was coded as "point prevalence." "Continuous" was used when a study reported abstinence based on whether study subjects were continuously abstinent from tobacco use since their quit day. "Unknown" was used when it was not possible to discern from the study report whether the authors used a point prevalence or continuous measure for abstinence.

As in the original guideline, a point prevalence outcome measure (1-week point prevalence, when available) rather than continuous abstinence was used as the chief outcome variable. Point prevalence was preferred for several reasons. First, among the 180 randomized controlled trials available for meta-analysis, the majority presented their primary outcome data as point prevalence. Second, continuous abstinence data underestimate the percentage of individuals who are abstinent at particular follow-up time points. They might, therefore, suggest that the likelihood of cessation is lower than in actuality. Finally, most relapse begins early in a quit attempt and persists. A point prevalence measure taken at 5 months would certainly capture the great majority of those relapse events. Therefore, wherever possible, 1-week point prevalence abstinence data were used. If point prevalence data were not available, the preferred alternative was continuous abstinence data. Data of an unknown or unspecified nature were used otherwise.

Meta-Analytic Techniques. The principal analytic technique used in this guideline was meta-analysis. This statistical technique estimates the impact of a treatment or variable across a set of related investigations. The primary meta-analytic model used in this and the original guideline was logistic regression using random effects modeling. The modeling was done at the level of the treatment arm, and study effects were treated as fixed. The panel methodologists chose to employ random effects modeling, assuming that both the subject populations and the treatment elements analyzed would vary from study to study (eg, "general problem-solving" counseling might be done somewhat differently at two different sites). Random effects modeling is well suited to accommodate such variation among studies.[38] The statistician used the EGRET Logistic Normal Model (Statistics and Epidemiology Research Corporation, *EGRET Reference Manual*, Revision 4, Seattle, 1993). A complete and detailed review of the meta-analytic methods used in the guideline can be found in the *Smoking Cessation Guideline Technical Report No. 18* available from AHRQ as AHCPR Publication No. 97-N004.

The meta-analytic technique assumed randomization of subjects to treatment conditions. Moreover, studies that randomized at another level (eg, clinician, clinic, etc) typically did not provide data on nonindependence. Therefore, only studies that randomized at the level of the subject were submitted to meta-analysis. To reduce the likelihood that this selection criterion would bias results, some test analyses were performed that included studies randomized by clinic or clinician. In all cases, these analyses were consistent with the results of studies using subject-based randomization.

The initial step in meta-analysis was the selection of studies that were relevant to the treatment characteristic being evaluated. After relevant studies were identified (eg, those that contained a self-help intervention if self-help treatments were being evaluated), panel staff reviewed the studies to ensure that they passed screening criteria. Some screening criteria were general (eg, study presents greater than 5 mo follow-up data), whereas other criteria were specific to the type of treatment characteristic evaluated (eg, in the analysis of clinicians, screening ensured that differences in type of clinician were not confounded by differences in use of pharmacotherapy). In most cases, there was no attempt to control for the effects of variables that were potentially correlated with an analyzed treatment dimension (eg, controlling for overall treatment intensity in the analysis of number of types of formats and number of types of clinicians).

The separate arms (treatment or control groups) in each study were then inspected to identify confounders that could compromise interpretation. Seriously confounded arms were excluded from analysis. Relevant characteristics of each arm were then coded to produce meaningful analytic comparisons. Criteria for performing a meta-analysis included: (1) the guideline panel judged the topic to be addressed in the meta-analysis as having substantial clinical importance, (2) at least two studies meeting selection criteria existed on the topic and the studies contained suitable within-study control or comparison conditions, and (3) there was acceptable inter-study homogeneity in the analyzed variable or treatment to permit meaningful inference (eg, an analyzed treatment was sufficiently similar across various studies so that combining studies was meaningful).

Limitations of Meta-Analytic Techniques. Several factors can compromise the internal validity of meta-analyses. For example, publication biases (particularly the tendency to publish only those studies with positive findings) may result in biased summary statistics. The complement to publication bias is the "file-drawer effect," in which negative or neutral findings are not submitted for publication. In addition, either the magnitude or the significance of the effects of meta-analyses may be influenced by factors such as the frequency with which treatments occurred in the data set, and by the extent to which treatments co-occurred with other treatments. All else being equal, a treatment that occurs infrequently in the data set is less likely to be found significant than a more frequently occurring treatment. Also, when two treatments co-occur frequently in the same groups of subjects, it is difficult to apportion statistically the impact of each. In addition, comparability biases can exist when substantially different groups or treatments are coded as being the same (eg, when treatments are similar only on a superficial attribute).

Stability of meta-analytic findings was determined with respect to only one population characteristic: that is whether patients sought cessation treatment ("self-selected") or whether treatment was delivered without the patient seeking it ("all-comers," as when cessation treatment occurred as an integral part of health care). Conducting separate meta-analyses in these different subject populations yielded very similar findings across a variety of treatment dimensions (eg, treatment format, treatment intensity). No other population characteristic (eg, years smoked, severity of dependence) was explored in meta-analyses.

Interpretation of Meta-analysis Results. The meta-analyses yielded logistic regression coefficients that were converted to odds ratios. The meaning or interpretation of an odds ratio can be seen most easily by means of an example depicted in a 2 × 2 table. Table 1 contains data showing the relation between maternal smoking and low birthweight in infants. Data are extracted from Hosmer and Lemeshow.[39] The odds of a low birthweight infant if the mother smokes are 30:44, or 0.68 to 1. The odds of a low birthweight infant if the mother does not smoke are 29:86, or 0.34 to 1. The odds ratio is thus (30/44)/(29/86) = 2.02 to 1. Therefore, the odds ratio can be seen roughly as the odds of an outcome on one variable, given a certain status on another variable(s). In the case above, the odds of a low birthweight infant is about double for women who smoke compared with those who do not.

Once odds ratios were obtained from meta-analyses, 95% confidence intervals (CIs) were estimated around the odds ratios. An odds ratio is only an estimate of a relation between variables. The 95% CI presents an estimate of the accuracy of the particular odds ratio obtained. If the 95% CI for a given odds ratio does not include "1," then the odds ratio represents a statistically significant effect at the 0.05 level. The CIs will generally not be perfectly symmetrical around an odds ratio because of the distributional properties of the odds ratio. Comparisons of the relative

Table 1. Relation Between Maternal Smoking and Low Birth Weight in Infants

		Maternal Smoking		
		Yes	No	Totals
Low Birth Weight	Yes	30	29	59
	No	44	86	130
Totals		74	115	189

Table 2. Meta-Analysis: Efficacy of and Estimated Abstinence Rates for Various Types of Format (n = 58 studies)

Format	Number of Arms	Estimated Odds Ratio (95% CI)	Estimated Abstinence Rate (95% CI)
No format	20	1.0	10.8
Self-help	93	1.2 (1.02, 1.3)	12.3 (10.9, 13.6)
Proactive telephone counseling	26	1.2 (1.1, 1.4)	13.1 (11.4, 14.8)
Group counseling	52	1.3 (1.1, 1.6)	13.9 (11.6, 16.1)
Individual counseling	67	1.7 (1.4, 2.0)	16.8 (14.7, 19.1)

CI = confidence interval

sizes of odds ratios is meaningful only for those odds ratios yielded by the same meta-analysis (listed in the same data table).

After computing the odds ratios and their CIs, the odds ratios were converted to abstinence percentages and their 95% CIs (based on reference category abstinence rates). Abstinence percentages indicate the estimated long-term abstinence rate achieved under the tested treatment or treatment characteristic. The abstinence percentage results are approximate estimates derived from the odds ratio data.[40] Therefore, they essentially duplicate the odds ratio results but are presented because their meaning may be clearer for some readers. Because the placebo/control abstinence percentage for a particular analysis is calculated exclusively from the studies included within that meta-analysis, these abstinence percentages vary across the different analyses.

How To Read the Data Tables. Table 2 depicts a table of results from one of the meta-analyses reported in this guideline. This table presents results from the analysis of the effects of different treatment formats on outcome (see Formats of Psychosocial Treatments in Chapter 6B). In this table, the comparison condition, or "reference group," for determining the impact of different treatment options, was smokers who had no contact with a provider. The "Estimated odds ratio" column reveals that treatment groups receiving self-help treatment had an odds ratio of 1.2. The odds ratio indicates a significant effect, because the lower boundary of the CI did not include "1." Proactive telephone counseling had an odds ratio of 1.2, and group counseling had an odds ratio of 1.3. Both of these are statistically significant because the lower bounds of their CIs do not include "1". Individual counseling had the largest odds ratio of 1.7. This odds ratio means that when a smoker receives individual counseling, he or she is more than one and one-half times more likely to remain abstinent than if he or she had received no counseling.

The column labeled "Estimated abstinence rate" shows the abstinence percentages for the various treatment formats. For instance, the reference group conditions (no contact) in the analyzed data set were associated with an abstinence rate of 10.8%. Consistent with the odds ratio data reviewed above, self-help treatments produced modest increases in abstinence rates (12.3%), proactive telephone counseling and group counseling produced somewhat larger increases in abstinence rates (13.1% and 13.9%, respectively), and individual counseling produced the largest increase (16.8%).

The total number of studies included in each meta-analysis is provided within the title of the corresponding table. A list of published articles used in each meta-analysis is available from AHRQ via the AHRQ Web site at www.ahrq.gov.

The column labeled "Number of arms" lists the number of treatment conditions or groups across all analyzed studies that contributed data to the various treatment format categories (eg, self-help treatment was provided in 93 treatment arms). Therefore, this column depicts the number of treatment conditions or groups relevant to each analyzed category. Frequently, the number of treatment groups or arms exceeds the number of studies included in a meta-analysis.

The outcome data in the tables include studies with "all-comers" (individuals who did not seek a treatment intervention) and "self-selected" populations, studies using point-prevalence and continuous abstinence end points, and studies with and without biochemical confirmation, except where otherwise described. Some meta-analyses (such as those evaluating pharmacotherapies) included predominantly studies with "self-selected" populations. In addition, in pharmacotherapy studies both experimental and control subjects typically received substantial counseling. Both of these factors tend to produce higher abstinence rates in reference or placebo subjects than are typically observed among self-quitters.

Strength of Evidence. Every recommendation made by the panel bears a strength-of-evidence rating that indicates the quality and quantity of empirical support for the recommendation. Each recommendation and its strength of evidence reflect consensus of the guideline panel. The three strength of evidence ratings are described below:

- **A** Multiple well-designed randomized clinical trials, directly relevant to the recommendation, yielded a consistent pattern of findings.
- **B** Some evidence from randomized clinical trials supported the recommendation, but the scientific support was not optimal. For instance, few randomized trials existed, the trials that did exist were somewhat inconsistent, or the trials were not directly relevant to the recommendation.

C Reserved for important clinical situations where the panel achieved consensus on the recommendation in the absence of relevant randomized controlled trials.

The availability of randomized clinical trials was not considered in economic recommendations. The existence of such trials was thought to be a less germane criterion for evaluating economic studies. In such cases, the strength of evidence is based primarily on the consistency of findings among different studies. Finally, the panel declined to make recommendations when there was no relevant evidence or the evidence was too weak or inconsistent to support a recommendation.

Caveats Regarding Recommendations. The reader should note some caveats regarding guideline recommendations. First, an absence of studies should not be confused with proven lack of efficacy. In certain situations, there was little direct evidence regarding the efficacy of some treatments, and in these cases the panel usually rendered no opinion. Second, even when there were enough studies to perform a meta-analysis, a nonsignificant result does not prove inefficacy. Rather, nonsignificance merely indicates that efficacy was not demonstrated given the data available.

The emphasis of this guideline was to identify efficacious interventions, not to rank-order interventions in terms of efficacy. The panel chose not to emphasize comparisons among efficacious interventions, for several reasons. First, the most important goal of the analytic process was to identify all efficacious interventions. Second, selection or use of particular intervention techniques or strategies is usually a function of practical factors: patient preference, time available, training of the clinician, cost, and so on. The panel believed clinicians should choose the most appropriate intervention from among the efficacious interventions, given existing circumstances. An excessive emphasis on relative efficacy might discourage clinicians from using interventions that have a small, but reliable, impact on smoking cessation. Finally, data were often inadequate or unavailable to make adequate statistical comparisons of different types of interventions. For example, there were insufficient studies testing head-to-head comparisons of the different pharmacotherapies to allow a rank-ordering of the different pharmacotherapies.

Despite a lack of emphasis on the rank-ordering of interventions, some interventions were so superior to control or no-treatment conditions that the panel clearly identified them as superior to another intervention. For instance, although minimal person-to-person contact can increase smoking abstinence rates over no-treatment conditions, there is little doubt that longer person-to-person interventions have greater impact.

External Review of the Guideline. The panel and AHRQ invited 155 outside reviewers to review the draft of the 1996 guideline. In addition, AHRQ placed a notice in the *Federal Register* inviting individuals to review and comment on this original draft guideline. A total of 71 reviewers provided comments. Panel and consortium members invited 175 outside reviewers to review the updated guideline. A total of 70 provided comments. Peer reviewers included clinicians, health care administrators, social workers, counselors, health educators, researchers, consumers, key personnel at selected federal agencies, and others. Reviewers were asked to evaluate the guideline based on five criteria: validity, reliability, clarity, clinical applicability, and utility. Comments of the peer reviewers were incorporated into the guideline when appropriate.

Guideline Products

Accompanying the original guideline were four products intended to aid in the dissemination and translation of the guideline's evidence-based recommendations. These products were intended to address consumers as well as the three target audiences: primary care clinicians, specialists, and health care administrators, insurers, and purchasers. These products were: (1) *You Can Quit Smoking. Consumer Guide*, (2) *Helping Smokers Quit. A Guide for Primary Care Clinicians*, (3) *Smoking Cessation: Information for Specialists. Quick Reference Guide*, and (4) *Smoking Cessation, A Systems Approach: A Guide for Health Care Administrators, Insurers, Managed Care Organizations, and Purchasers*. Similar products will disseminate the recommendations of the updated guideline.

Organization of the Guideline Update

This updated guideline is divided into eight chapters that reflect the major components of tobacco dependence treatment (Fig. 2):

Chapter 1, Overview and Methods, provides an overview and rationale for the updated guideline, as well as a detailed description of the methodology used to review the scientific literature and develop the original and updated guidelines.

Chapter 2, Assessment of Tobacco Use, establishes the importance of determining the tobacco use status of every patient at every visit.

Chapter 3, Brief Clinical Interventions, is intended to provide clinicians with guidance as they use brief interventions to treat tobacco users willing to quit, tobacco users unwilling to make a quit attempt at this time, and tobacco users who have recently quit.

A. For the Patient Willing To Quit, provides brief clinical approaches to assist patients in quit attempts.

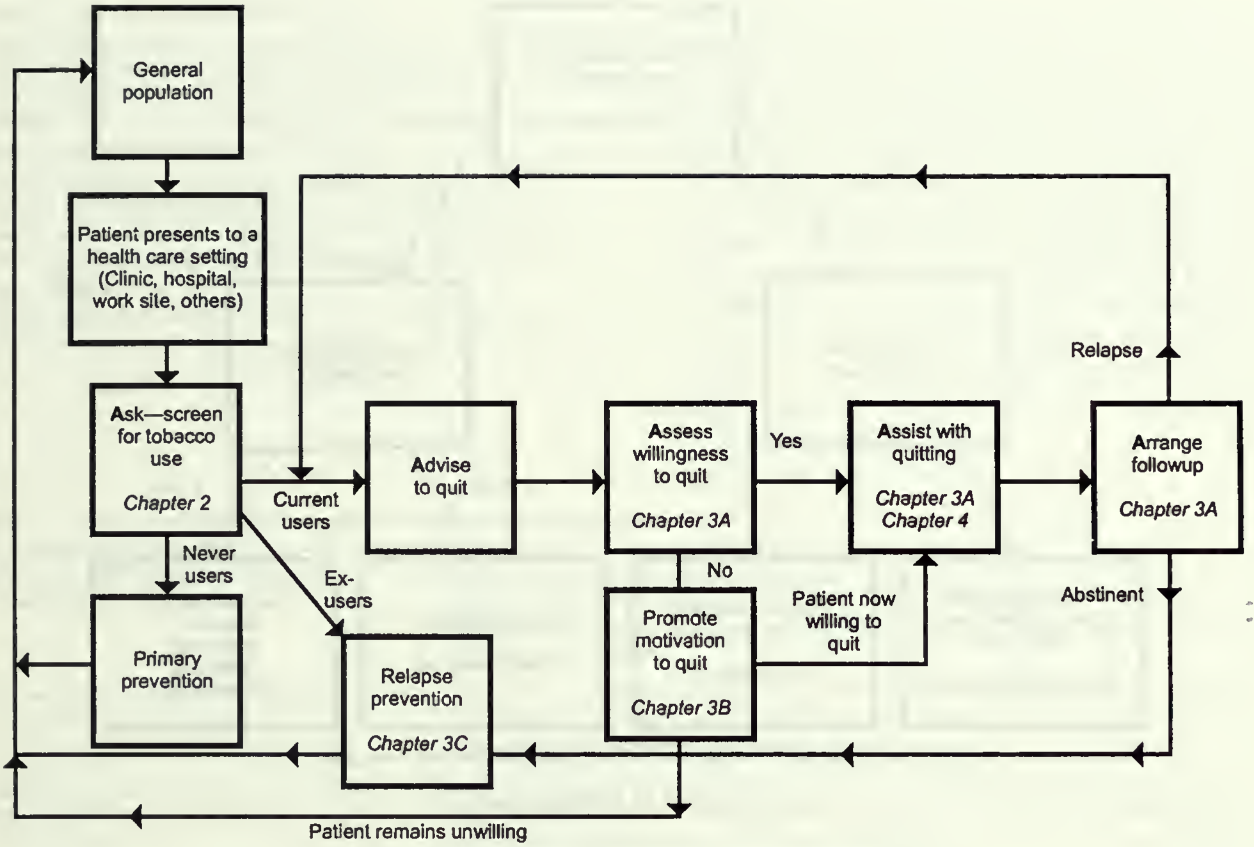


Fig. 2. Model for treatment of tobacco use and dependence.

B. For the Patient Unwilling To Quit, provides brief clinical approaches designed to motivate the patient to make a quit attempt at this time.

C. For the Patient Who Has Recently Quit, provides clinicians with brief strategies designed to reinforce an ex-tobacco user's commitment to stay tobacco-free.

Chapter 4, Intensive Clinical Interventions, provides clinicians with more intensive strategies to treat tobacco users.

Chapter 5, Systems Interventions: Relevance to Health Care Administrators, Insurers, and Purchasers, is directed at health care administrators, insurers, purchasers, and other decision-makers who can affect health care systems. This chapter provides these decision-makers with strategies to modify health care systems to improve the delivery of tobacco treatment services.

Chapter 6, Evidence, presents the evidentiary basis for the updated guideline recommendations.

A. Screening and Assessment, provides recommendations and analysis results regarding screening for tobacco use and specialized assessment.

B. Treatment Structure and Intensity, provides recommendations and analysis results regarding advice, intensity of clinical interventions, and type of clinician, format, and follow-up procedures.

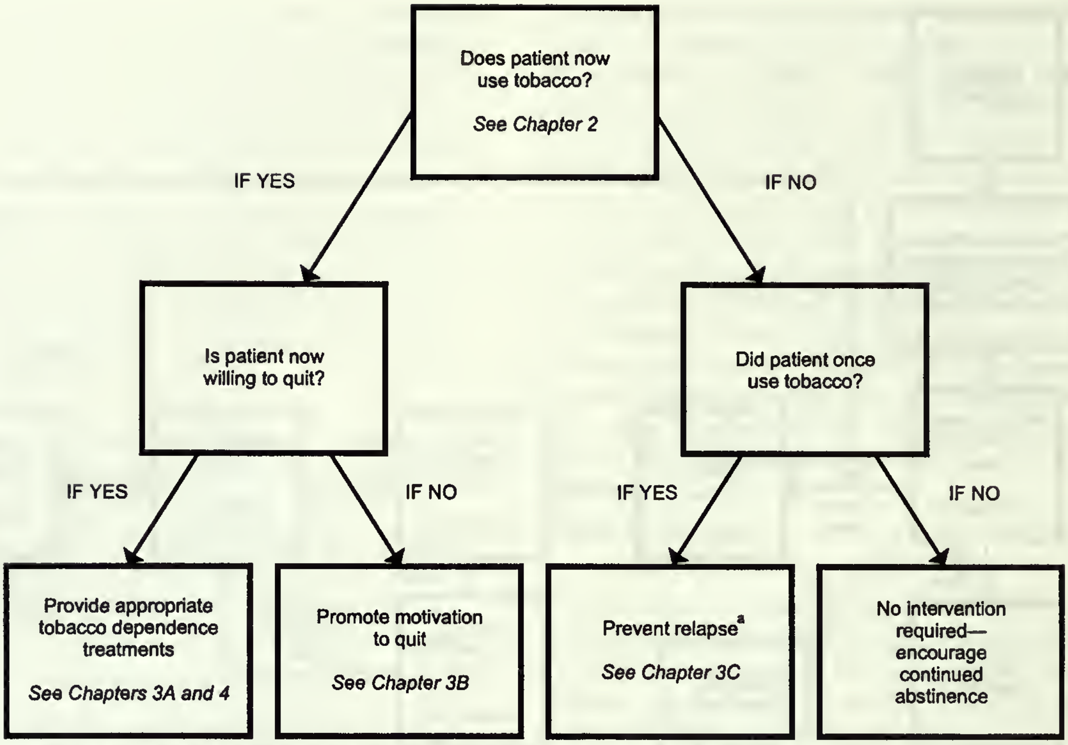
C. Treatment Elements, provides recommendations and analysis results regarding types of counseling and behavioral therapies and pharmacotherapy

Chapter 7, Special Populations, provides information on specific populations such as women, pregnant tobacco users, racial and ethnic minorities, hospitalized patients, older adults, tobacco users with other chemical dependencies, and children and adolescents.

Chapter 8, Special Topics, provides specific information on topics not otherwise addressed in the updated guideline such as weight gain associated with tobacco cessation, noncigarette tobacco products, clinician training, and reimbursement for tobacco cessation treatment.

2. Assessment of Tobacco Use

At least 70% of smokers see a physician each year, and more than 50% see a dentist.[41–43] Other smokers see physician assistants, nurse practitioners, nurses, physical and occupational therapists, pharmacists, and other clinicians. Therefore, all clinicians, particularly physicians and dentists, are uniquely poised to intervene with patients who use tobacco. Moreover, 70% of smokers report wanting to quit.[3] Finally, smokers cite a physician's advice to quit as


Does patient now use tobacco?
See Chapter 2
IF YES
IF NO
Is patient now willing to quit?
Did patient once use tobacco?
IF YES
IF NO
IF YES
IF NO
Provide appropriate tobacco dependence treatments
See Chapters 3A and 4
Promote motivation to quit
See Chapter 3B
Prevent relapse[a]
See Chapter 3C
No intervention required—encourage continued abstinence

[a] Relapse prevention interventions are not necessary in the case of the adult who has not used tobacco for many years.

Fig. 3. Algorithm for treating tobacco use.

an important motivator for attempting to stop smoking.[44–46] These data suggest that most smokers are interested in quitting, clinicians are frequently in contact with smokers, and clinicians have high credibility with smokers.

Unfortunately, clinicians are not capitalizing on this unique opportunity. More than one third of current smokers report never having been asked about their smoking status or urged to quit.[11,47] Moreover, a population-based survey found that less than 15% of smokers who saw a physician in the past year were offered assistance, and only 3% had a follow-up appointment to address tobacco use.[48] Fewer still have received specific advice on how to quit smoking successfully. This guideline clearly identifies empirically validated tobacco treatment strategies to spur clinicians, tobacco treatment specialists, and administrators to intervene effectively with patients who use tobacco.

The first step in treating tobacco use and dependence is to identify tobacco users. As the data analysis in Chapter 6 shows, the identification of smokers itself increases rates of clinician intervention. Effective identification of tobacco use status not only opens the door for successful interventions (eg, physician advice), but also it guides clinicians to identify appropriate interventions based on patients' tobacco use status and willingness to quit.

Screening for current or past tobacco use will result in four possible responses: (1) the patient uses tobacco and is now willing to make a quit attempt, (2) the patient uses tobacco but is not now willing to make a quit attempt, (3) the patient once used tobacco but has since quit, or (4) the patient never regularly used tobacco. This clinical practice guideline is organized to provide the clinician with simple but effective interventions for all of these patients (Fig. 3).

3. Brief Clinical Interventions

Background

This section of the guideline presents specific strategies to guide clinicians providing brief interventions. These brief interventions can be provided by any clinician but are most relevant to primary care clinicians (physicians, nurses, dentists, hygienists, respiratory therapists, pharmacists, etc) who see a wide variety of patients and who are bound by time constraints. These strategies are based on the evidence described in Chapters 6, 7, and 8, as well as on panel opinion. Guideline analysis suggests that a wide variety of clinicians can implement these brief strategies effectively. The goals of these strategies are clear: to change clinical culture and practice patterns to ensure that every patient who uses tobacco is identified and offered treatment. The strategies underscore a central theme: it is essential to

Table 3. The "5 A's" for Brief Intervention

- Ask about tobacco use. Identify and document tobacco use status for every patient at every visit. (Brief Strategy A1)
- Advise to quit. In a clear, strong and personalized manner urge every tobacco user to quit. (Brief Strategy A2)
- Assess willingness to make a quit attempt. Is the tobacco user willing to make a quit attempt at this time? (Brief Strategy A3)
- Assist in quit attempt. For the patient willing to make a quit attempt, use counseling and pharmacotherapy to help him or her quit. (Brief Strategy A4)
- Arrange follow-up. Schedule follow-up contact, preferably within the first week after the quit date. (Brief Strategy A5)

provide at least a brief intervention to all tobacco users at each clinical visit. Several observations are relevant to this theme. First, institutional changes in clinical practice are necessary to ensure that all patients who use tobacco are identified for intervention (see Chapter 5, Systems Interventions: Relevance to Health Care Administrators, Insurers, and Purchasers). Second, the compelling time limits on primary care physicians in the United States today[49] (median visit = 12–16 min)[50] often require brief interventions, although more intensive interventions would produce greater success. Third, although many smokers are reluctant to seek intensive cessation programs,[51] they nevertheless can receive a brief intervention every time they visit a clinician.[22]

Table 4. Clinical Guidelines for Prescribing Pharmacotherapy for Smoking Cessation

Who should receive pharmacotherapy for smoking cessation?	All smokers trying to quit, except in the presence of special circumstances. Special consideration should be given before using pharmacotherapy with selected populations: those with medical contraindications, those smoking fewer than 10 cigarettes/day, pregnant/breastfeeding women, and adolescent smokers.
What are the first-line pharmacotherapies recommended in this guideline?	All five of the United States Food and Drug Administration (FDA)-approved pharmacotherapies for smoking cessation are recommended, including bupropion SR, nicotine gum, nicotine inhaler, nicotine nasal spray, and nicotine patch.
What factors should a clinician consider when choosing among the 5 first-line pharmacotherapies?	Because of the lack of sufficient data to rank-order these 5 medications, choice of a specific first-line pharmacotherapy must be guided by factors such as clinician familiarity with the medications, contraindications for selected patients, patient preference, previous patient experience with a specific pharmacotherapy (positive or negative), and patient characteristics (eg, history of depression, concerns about weight gain).
Are pharmacotherapeutic treatments appropriate for lighter smokers (eg, 10–15 cigarettes/day)?	If pharmacotherapy is used with lighter smokers, clinicians should consider reducing the dose of first-line nicotine replacement therapy pharmacotherapies. No adjustments are necessary when using bupropion SR.
What second-line pharmacotherapies are recommended in this guideline?	Clonidine and nortriptyline.
When should second-line agents be used for treating tobacco dependence?	Consider prescribing second-line agents for patients unable to use first-line medications because of contraindications or for patients for whom first-line medications are not helpful. Monitor patients for the known adverse effects of second-line agents.
Which pharmacotherapies should be considered with patients particularly concerned about weight gain?	Bupropion SR and nicotine replacement therapies, in particular nicotine gum, have been shown to delay, but not prevent, weight gain.
Are there pharmacotherapies that should be especially considered in patients with a history of depression?	Bupropion SR and nortriptyline appear to be effective with this population.
Should nicotine replacement therapies be avoided in patients with a history of cardiovascular disease?	No. The nicotine patch in particular is safe and has been shown not to cause adverse cardiovascular effects.
May tobacco dependence pharmacotherapies be used long-term (eg, 6 months or more)?	Yes. This approach may be helpful with smokers who report persistent withdrawal symptoms during the course of pharmacotherapy or who desire long-term therapy. A minority of individuals who successfully quit smoking use ad libitum nicotine replacement therapy medications (gum, nasal spray, inhaler) long-term. The use of these medications long-term does not present a known health risk. Additionally, the FDA has approved the use of bupropion SR for a long-term maintenance indication.
May pharmacotherapies ever be combined?	Yes. There is evidence that combining the nicotine patch with either nicotine gum or nicotine nasal spray increases long-term abstinence rates over those produced by a single form of nicotine replacement therapy.

Action	**Strategies for Implementation**
Implement an office-wide system that ensures that for EVERY patient at EVERY clinic visit tobacco-use status is queried and documented. Repeated assessment is *not* necessary in the case of the adult who has never used tobacco or has not used tobacco for many years and for whom this information is clearly documented in the medical record.	Expand the vital signs to include tobacco use or use an alternative universal identification system. Vital Signs Blood Pressure: ____________________ Pulse: ________ Weight: ________ Temperature: ____________________ Respiratory Rate: ____________________ Tobacco Use: Current Former Never *(circle one)* Alternatives to expanding the vital signs are to place tobacco-use status stickers on all patient charts or to indicate tobacco use status using electronic medical records or computer reminder systems.

Brief Strategy A1. Ask: Systemically identify all tobacco users at every visit.

Action	**Strategies for Implementation**
In a *clear*, *strong*, and *personalized* manner, urge every tobacco user to quit.	Advice should be: • *Clear*: "I think it is important for you to quit smoking now and I can help you." "Cutting down while you are ill is not enough." • *Strong*: "As your clinician, I need you to know that quitting smoking is the most important thing you can do to protect your health now and in the future. The clinic staff and I will help you." • *Personalized*: Tie tobacco use to current health/illness, and/or its social and economic costs, motivation level/readiness to quit, and/or the impact of tobacco use on children and others in the household.

Brief Strategy A2. Advise: Strongly urge all tobacco users to quit.

This chapter is divided into three sections to guide brief clinician interventions with three types of patients: (A) current tobacco users now willing to make a quit attempt, (B) current tobacco users unwilling at this time to make a quit attempt, and (C) former tobacco users who have recently quit. Adults who have never used tobacco or who have been abstinent for an extended period do not require intervention. The clinician may congratulate them on their status and encourage them to maintain their tobacco-free lifestyle.

A. For the Patient Willing To Quit

Given that so many tobacco users visit a primary care clinician each year, it is important that these clinicians be prepared to intervene with tobacco users who are willing to quit. The five major steps (the "5 A's") to intervention in the primary care setting are listed in Table 3. It is important for the clinician to *ask* the patient if he or she uses tobacco (Brief Strategy A1), *advise* him or her to quit (Brief Strategy A2), *assess* willingness to make a quit attempt (Brief Strategy A3), *assist* him or her in making a quit attempt (Brief Strategy A4), and *arrange* for follow-up contacts to prevent relapse (Brief Strategy A5). The strategies are designed to be brief, requiring 3 minutes or less of direct clinician time. Office systems that institutionalize tobacco use assessment and intervention will greatly foster the adoption of these strategies (see Chapter 5). Finally, these strategies are consistent with those produced by the NCI[13,52] and the American Medical Association,[27] as well as others.[26,53,54]

Action	Strategies for Implementation
Ask every tobacco user if he or she is willing to make a quit attempt at this time (eg, within the next 30 days).	Assess patient's willingness to quit: • If the patient is willing to make a quit attempt at this time, provide assistance (see Chapter 3A, Brief Strategy A4). • If the patient will participate in an intensive treatment, deliver such a treatment or refer to an intensive intervention (see Chapter 4). • If the patient clearly states he or she is unwilling to make a quit attempt at this time, provide a motivational intervention (see Chapter 3B). • If the patient is a member of a special population (eg, adolescent, pregnant smoker, racial/ethnic minority), consider providing additional information (see Chapter 7).

Brief Strategy A3. Assess: Determine willingness to make a quit attempt.

In addition to counseling, all smokers making a quit attempt should receive pharmacotherapy, except in the presence of special circumstances. Table 4 shows guidelines for prescribing pharmacotherapy for smoking cessation.

B. For the Patient Unwilling To Quit

Promoting the Motivation To Quit. All patients entering a health care setting should have their tobacco use status assessed routinely. Clinicians should advise all tobacco users to quit and then assess the patient's willingness to make a quit attempt. For patients not ready to make a quit attempt at this time, clinicians should use a brief intervention designed to promote the motivation to quit.

Patients unwilling to make a quit attempt during a visit may lack information about the harmful effects of tobacco, may lack the required financial resources, may have fears or concerns about quitting, or may be demoralized because of previous relapse.[55] Such patients may respond to a motivational intervention that provides the clinician an opportunity to educate, reassure, and motivate, such as the motivational intervention built around the "5 R's": *relevance*, *risks*, *rewards*, *roadblocks*, and *repetition*. Clinical components of the "5 R's" are shown in Brief Strategy B, Page 1218. Motivational interventions are most likely to be successful when the clinician is *empathic*, *promotes patient autonomy* (eg, choice among options), *avoids arguments*, and *supports the patient's self-efficacy* (eg, by identifying previous successes in behavior change efforts).[56–58]

C. For the Patient Who Has Recently Quit

Preventing Relapse. Because of the chronic relapsing nature of tobacco dependence, clinicians should provide brief effective relapse prevention treatment. When clinicians encounter a patient who has quit tobacco use recently, they should reinforce the patient's decision to quit, review the benefits of quitting, and assist the patient in resolving any residual problems arising from quitting. Although most relapse occurs early in the quitting process,[59,60] some relapse occurs months or even years after the quit date.[31,61] Therefore, clinicians should engage in relapse prevention interventions even with former tobacco users who no longer consider themselves actively engaged in the quitting process. Relapse prevention interventions are especially important soon after quitting and can be delivered by means of either scheduled clinic visits, telephone calls, or any time the clinician encounters an ex-tobacco user. A systematic, institutionalized mechanism to identify recent quitters and contact them is essential to deliver relapse prevention messages effectively.

Relapse prevention interventions can be divided into two categories: minimal practice and prescriptive interventions.

Minimal Practice Interventions. Minimal practice relapse prevention interventions should be part of every encounter with a patient who has quit recently (Brief Strategy C1, Page 1219). These interventions are appropriate for most recent quitters and can be addressed briefly during a coincident clinic visit or a scheduled follow-up visit. Because most relapse occurs within the first 3 months after quitting, relapse prevention is especially appropriate during this period.[62,63] In addition, strategies designed to reduce relapse should be included in the initial preparation for a quit attempt (see Chapter 3A, Brief Strategy A4. Assist: Aid the patient in quitting, and Chapter 3B, Brief Strategy B. Enhancing motivation to quit tobacco—the "5 R's"). Finally, encourage patients to report difficulties promptly (eg, lapses, depression, medication side-effects) while continuing efforts to quit.

Prescriptive Interventions. Prescriptive relapse prevention components are individualized based on information obtained about problems the patient has encountered in

Action	Strategies for Implementation
Help the patient with a quit plan.	A patient's preparations for quitting: • *Set* a quit date: Ideally, the quit date should be within 2 weeks. • *Tell* family, friends, and coworkers about quitting, and request understanding and support. • *Anticipate* challenges to planned quit attempt, particularly during the critical first few weeks. These include nicotine withdrawal symptoms. • *Remove* tobacco products from your environment. Prior to quitting, avoid smoking in places where you spend a lot of time (eg, work, home, car).
Provide practical counseling (problem-solving/ skills training).	*Abstinence*: Total abstinence is essential. "Not even a single puff after the quit date." *Past quit experience*: Identify what helped and what hurt in previous quit attempts. *Anticipate triggers or challenges in upcoming attempt*: Discuss challenges/triggers and how patient will successfully overcome them. *Alcohol*: Since alcohol can cause relapse, the patient should consider limiting/abstaining from alcohol while quitting. *Other smokers in the household*: Quitting is more difficult when there is another smoker in the household. Patients should encourage housemates to quit with them or not smoke in their presence.
Provide intra-treatment social support.	Provide a supportive clinical environment while encouraging the patient in his or her quit attempt. "My office staff and I are available to assist you." (See Table 22)
Help patient obtain extra-treatment social support.	Help patient develop social support for his or her quit attempt in his or her environments outside of treatment. "Ask your spouse/partner, friends, and coworkers to support you in your quit attempt." (See Table 23)
Recommend the use of approved pharmacotherapy, except in special circumstances.	Recommend the use of pharmacotherapies found to be effective in this guideline (see Table 4 for clinical guidelines and Tables 33-39 for specific instructions and precautions). Explain how these medications increase smoking cessation success and reduce withdrawal symptoms. The first-line pharmacotherapy medications include: bupropion SR, nicotine gum, nicotine inhaler, nicotine nasal spray, and nicotine patch.
Provide supplementary materials.	*Sources*: Federal agencies, nonprofit agencies, or local/state health departments *Type*: Culturally/racially/educationally/age appropriate for the patient. *Location*: Readily available at every clinician's workstation.

Brief Strategy A4. Assist: Aid the patient in quitting.

maintaining abstinence (Brief Strategy C2, Page XX). These more intensive relapse prevention interventions may be delivered during a dedicated follow-up contact (in person or by telephone) or through a specialized clinic or program.

4. Intensive Clinical Interventions

Background

Intensive tobacco dependence treatment can be provided by any suitably trained clinician who has the resources available to give intensive interventions. Based on the evidence in Chapter 6, it has been shown that more intensive tobacco dependence treatment is more effective than brief treatment. Also, it should be noted that intensive interventions are appropriate for any tobacco user willing to participate in them. There is no evidence that the efficacy or cost-effectiveness of intensive interventions is limited to a subpopulation of tobacco users (eg, heavily dependent smokers).[64]

In many cases, intensive tobacco dependence interventions are provided by clinicians who specialize in the treatment of tobacco dependence. Such specialists are not de-

Action	Strategies for Implementation
Schedule follow-up contact, either in person or via telephone.	*Timing*: Follow-up contact should occur soon after the quit date, preferably during the first week. A second follow-up contact is recommended within the first month. Schedule further follow-up contacts as indicated.
	Actions during follow-up contact: Congratulate success. If tobacco use has occurred, review circumstances and elicit recommitment to total abstinence. Remind patient that a lapse can be used as a learning experience. Identify problems already encountered and anticipate challenges in the immediate future. Assess pharmacotherapy use and problems. Consider use or referral to more intensive treatment (see Chapter 4).

Brief Strategy A5. Arrange: Schedule followup contact.

fined by their professional affiliation or by the field in which they trained. Rather, specialists view tobacco dependence treatment as a primary professional role. Specialists possess the skills, knowledge, and training to provide efficacious interventions across a range of intensities, and are often affiliated with programs offering intensive treatment interventions or services (programs with staff dedicated to tobacco interventions, where treatment involves multiple counseling sessions, and so on). In addition to offering intensive treatments, specialists often conduct research on tobacco dependence and its treatment.

As noted above, there is substantial evidence that intensive interventions produce higher success rates than do less intensive interventions (as indicated by several findings of this guideline). In addition, the tobacco dependence interventions offered by specialists represent an important treatment resource for patients who do not receive tobacco dependence treatment from their primary care clinician.

Although the specialist contributes greatly to tobacco treatment efforts, constraints limit the impact of the specialist's service delivery activities. For example, only a minority of smokers participate in the intensive programs typically offered by specialists.[51,65] This suggests that, in the future, the specialist may contribute to tobacco treatment efforts through activities such as:

- Serving as a resource to nonspecialists who offer tobacco dependence services as part of general health care delivery. This might include training nonspecialists in counseling strategies, providing consultation on difficult cases and for inpatients, and providing specialized assessment services.
- Developing and evaluating changes in office/clinic procedures that increase the rates at which tobacco users are identified and treated.
- Conducting evaluation research to determine the effectiveness of ongoing tobacco dependence treatment activities in relevant institutional settings.
- Developing and evaluating innovative treatment strategies that may increase the effectiveness and utilization of tobacco dependence treatments.

Strategies for Intensive Tobacco Dependence Intervention

Table 5 highlights guideline findings based on analyses found in Chapter 6, as well as on panel opinion, that seem particularly relevant to the implementation of intensive treatment programs. The findings from Table 5 lead to the development of an intensive treatment strategy (see Intensive Strategy. Components of an intensive intervention, Page 1221). Of course, implementation of this strategy depends on factors such as resource availability and time constraints.

5. Systems Interventions. Relevance to Health Care Administrators, Insurers, and Purchasers

Background

Traditionally, efforts to increase tobacco intervention in the health care setting have targeted the individual clinician. Such a restricted focus is no longer appropriate. Two considerations argue for expanding the scope of tobacco intervention efforts. First, efforts directed at the individual clinician have yielded disappointing results. For instance, national data suggest that, in a given visit with a clinician, more than one-third of smokers are not advised and assisted with cessation.[11,14,41] Second, an increasing number of Americans today receive their health care in managed care settings. As a consequence, agents such as health system administrators, insurers, and health care purchasers now play a significant role in the health care of most Americans. For example, managed care organizations and other insurers influence medical care through restrictive formularies, performance feedback to clinicians, and marketing approaches that prompt patient demand for particular services.

The influence of health care system administrators, insurers, and purchasers could, in theory, be used to encourage and support the consistent and effective identification and treatment of tobacco users. These agents could craft and implement supportive systems, policies, and environ-

Relevance	Encourage the patient to indicate why quitting is personally relevant, being as specific as possible. Motivational information has the greatest impact if it is relevant to a patient's disease status or risk, family or social situation (eg., having children in the home), health concerns, age, gender, and other important patient characteristics (eg., prior quitting experience, personal barriers to cessation).
Risks	The clinician should ask the patient to identify potential negative consequences of tobacco use. The clinician may suggest and highlight those that seem most relevant to the patient. The clinician should emphasize that smoking low-tar/low-nicotine cigarettes or use of other forms of tobacco (eg., smokeless tobacco, cigars, and pipes) will not eliminate these risks. Examples of risks are: • *Acute risks:* Shortness of breath, exacerbation of asthma, harm to pregnancy, impotence, infertility, increased serum carbon monoxide. • *Long-term risks:* Heart attacks and strokes, lung and other cancers (larynx, oral cavity, pharynx, esophagus, pancreas, bladder, cervix), chronic obstructive pulmonary diseases (chronic bronchitis and emphysema), long-term disability and need for extended care. • *Environmental risks:* Increased risk of lung cancer and heart disease in spouses; higher rates of smoking by children of tobacco users; increased risk for low birth weight, SIDS, asthma, middle ear disease, and respiratory infections in children of smokers.
Rewards	The clinician should ask the patient to identify potential benefits of stopping tobacco use. The clinician may suggest and highlight those that seem most relevant to the patient. Examples of rewards follow: • Improved health. • Food will taste better. • Improved sense of smell. • Save money. • Feel better about yourself. • Home, car, clothing, breath will smell better. • Can stop worrying about quitting. • Set a good example for children. • Have healthier babies and children. • Not worry about exposing others to smoke. • Feel better physically. • Perform better in physical activities. • Reduced wrinkling/aging of skin.
Roadblocks	The clinician should ask the patient to identify barriers or impediments to quitting and note elements of treatment (problem-solving, pharmacotherapy) that could address barriers. Typical barriers might include: • Withdrawal symptoms. • Fear of failure. • Weight gain. • Lack of support. • Depression. • Enjoyment of tobacco.
Repetition	The motivational intervention should be repeated every time an unmotivated patient visits the clinic setting. Tobacco users who have failed in previous quit attempts should be told that most people make repeated quit attempts before they are successful.

Brief Strategy B. Enhancing motivation to quit tobacco: The "5 R's".

mental prompts that render tobacco use treatment an integral part of health care. Indeed, research clearly shows that systems-level change can reduce smoking prevalence among enrollees of managed health care plans.[66]

Unfortunately, as a society we have not capitalized on the opportunity to use today's health care organizations, insurers, and purchasers to combat tobacco use. For example, treatments for tobacco use (both pharmacotherapy and counseling) are not consistently provided as paid services for subscribers of health insurance packages.[67,68] A recent survey found that only 22 states provided Medicaid coverage for tobacco dependence treatment.[69] Another survey demonstrated that only 39% of managed care organizations had partially implemented the recommendations

These interventions should be part of every encounter with a patient who has quit recently:

Every ex-tobacco user undergoing relapse prevention should receive congratulations on any success and strong encouragement to remain abstinent.

When encountering a recent quitter, use open-ended questions designed to initiate patient problem-solving (eg, How has stopping tobacco use helped you?). The clinician should encourage the patients' *active* discussion of the topics below:

- The benefits, including potential health benefits, the patient may derive from cessation.
- Any success the patient has had in quitting (duration of abstinence, reduction in withdrawal, etc.)
- The problems encountered or anticipated threats to maintaining abstinence (eg, depression, weight gain, alcohol, other tobacco users in the household).

Brief Strategy C1. Components of minimal practice relapse prevention.

During prescriptive relapse prevention, a patient might identify a problem that threatens his or her abstinence. Specific problems likely to be reported by patients and potential responses follow:

Problem	**Responses**
Lack of support for cessation	Schedule follow-up visits or telephone calls with the patient. Help the patient identify sources of support within his or her environment. Refer the patient to an appropriate organization that offers cessation counseling or support.
Negative mood or depression	If significant, provide counseling, prescribe appropriate medications, or refer the patient to a specialist.
Strong or prolonged withdrawal symptoms	If the patient reports prolonged craving or other withdrawal symptoms, consider extending the use of an approved pharmacotherapy or adding/combining pharmacologic medications to reduce strong withdrawal symptoms.
Weight gain	Recommend starting or increasing physical activity; discourage strict dieting. Reassure the patient that some weight gain after quitting is common and appears to be self-limiting. Emphasize the importance of a healthy diet. Maintain the patient on pharmacotherapy known to delay weight gain (eg, bupropion SR, nicotine replacement therapies, particularly nicotine gum). Refer the patient to a specialist or program.
Flagging motivation/feeling deprived	Reassure the patient that these feelings are common. Recommend rewarding activities. Probe to ensure that the patient is not engaged in periodic tobacco use. Emphasize that beginning to smoke (even a puff) will increase urges and make quitting more difficult.

Brief Strategy C2. Components of prescriptive relapse prevention.

within the original guideline, and only 9% had fully implemented the recommendations.[70] This lack of coverage is puzzling given that studies have shown that physician advice to quit is at least as cost-effective as several other preventive medical practices, including the treatment of mild or moderate hypertension or high cholesterol.[71,72] These and other findings resulted in the addition of a new objective to the national health promotion and disease prevention objectives for the year 2000 (*Healthy People 2000*):

- Increase to 100% the proportion of health plans that offer treatment of nicotine addiction (eg, tobacco use cessation counseling by health care providers, tobacco use cessation classes, prescriptions for nicotine replacement therapies, and/or other cessation services).[73]

Table 5. Findings Relevant to Intensive Interventions

- There is a strong dose-response relation between counseling intensity and cessation success. In general, the more intense the treatment intervention, the greater the rate of smoking cessation. Treatments may be made more intense by increasing (A) the length of individual treatment sessions and (B) the number of treatment sessions.
- Many different types of providers (physicians, nurses, dentists, psychologists, pharmacists, etc) are effective in increasing rates of tobacco cessation, and involving multiple types of providers may enhance abstinence rates.
- Proactive telephone calls and individual and group counseling are effective tobacco cessation formats.
- Particular types of counseling and behavioral therapies are especially effective. Practical counseling (problem-solving/skills-training approaches), and the provision of intra-treatment and extra-treatment social support are associated with significant increases in abstinence rates, as are aversive smoking techniques (eg, rapid smoking).
- Pharmacotherapies such as bupropion SR or nicotine replacement therapies consistently increase abstinence rates. Therefore, their use should be encouraged for all quitters, but special consideration is required with some populations (eg, pregnant smokers, adolescents).
- Tobacco dependence treatments are effective across diverse populations (eg, populations varying on gender, age, and ethnicity).

This objective has been modified in *Healthy People 2010* to state:

- Increase insurance coverage of evidence-based treatment for nicotine dependence to 100%.[74].

In sum, without supportive systems, policies, and environmental prompts, the individual clinician will likely not assess and treat tobacco use consistently. Therefore, just as clinicians must assume responsibility to treat their patients for tobacco use, so must health care administrators, insurers, and purchasers assume responsibility to craft policies, provide resources, and display leadership that results in consistent and effective tobacco use treatment.

Cost-Effectiveness of Smoking Cessation Interventions

Smoking cessation treatments are not only clinically effective, but they are economically defensible as well. It is vital that all three audiences targeted in this guideline recognize that smoking cessation treatments ranging from clinician advice to pharmacotherapy to specialist-delivered intensive programs are cost-effective in relation to other medical interventions.[64] Cost-effectiveness analyses have shown that smoking cessation treatments compare quite favorably with routine medical interventions such as the treatment of hypertension and hypercholesterolemia, and with other preventive interventions such as periodic mammography.[71,75–77] In fact, smoking cessation treatment has been referred to as the "gold standard" of preventive interventions.[78] Smoking cessation treatment remains highly cost-effective, even though a single application of any effective treatment for tobacco dependence may produce sustained abstinence in only a minority of smokers.

The cost-effectiveness of guideline recommendations for smoking cessation is addressed in detail in Chapter 8.

Recommendations for Health Care Administrators, Insurers, and Purchasers

Health care delivery administrators, insurers, and purchasers can promote the treatment of tobacco dependence through a systems approach. Purchasers (often corporations, companies, or other consortia that purchase health care benefits for a group of individuals) should make tobacco assessment and treatment a contractual obligation of the health care insurers and/or providers that sell services to them. In addition to improving the health of their employees or subscribers, mandating coverage for tobacco dependence treatment will result in lower rates of absenteeism[79] and lower utilization of health care resources.[80] Health care administrators and insurers must provide clinicians with assistance to ensure that institutional changes promoting tobacco dependence treatment are implemented universally and systematically. A number of institutional policies would facilitate these interventions such as:

- Implementing a tobacco-user identification system in every clinic (Systems Strategy 1).
- Providing education, resources, and feedback to promote provider intervention (Systems Strategy 2).
- Dedicating staff to provide tobacco dependence treatment and assessing the delivery of this treatment in staff performance evaluations (Systems Strategy 3).
- Promoting hospital policies that support and provide tobacco dependence services (Systems Strategy 4).
- Including tobacco dependence treatments (both counseling and pharmacotherapy) identified as effective in this guideline, as paid or covered services for all subscribers or members of health insurance packages (Systems Strategy 5).
- Reimbursing clinicians and specialists for delivery of effective tobacco dependence treatments and including these interventions among the defined duties of the clinicians (Systems Strategy 6).

These strategies are based on the evidence described in Chapters 6, 7, and 8, as well as on panel opinion.

6. Evidence

Background

The recommendations summarized in Chapters 2, 3, 4, and 5 are the result of a review and analysis of the extant tobacco cessation literature. The current chapter reports the results of this review and analysis, and describes the

Assessment	Assessments should ensure that tobacco users are willing to make a quit attempt using an intensive treatment program. Other assessments can provide information useful in counseling (eg, stress level, presence of comorbidity; see Chapter 6A, Specialized Assessment).
Program clinicians	Multiple types of clinicians are effective and should be used. One counseling strategy would be to have a medical/health care clinician deliver messages about health risks and benefits and deliver pharmacotherapy, and nonmedical clinicians deliver additional psychosocial or behavioral interventions.
Program intensity	Because of evidence of a strong dose-response relation, the intensity of the program should be: *Session length*: longer than 10 minutes. *Number of sessions*: 4 or more sessions. *Total contact time*: longer than 30 minutes.
Program format	Either individual or group counseling may be used. Proactive telephone counseling also is effective. Use of adjuvant self-help material is optional. Followup assessment intervention procedures should be used (see Chapter 6B).
Type of counseling and behavioral therapies	Counseling and behavioral therapies should involve practical counseling (problemsolving/skills-training) (see Table 21) and intra-treatment (see Table 22) and extra-treatment social support (see Table 23).
Pharmacotherapy	Every smoker should be encouraged to use pharmaco-therapies endorsed in this guideline, except in the presence of special circumstances. Special consideration should be given before using pharmacotherapy with selected populations (eg, pregnancy, adolescents) (see Table 4 for clinical guidelines and Tables 33-39 for specific instructions and precautions). The clinician should explain how these medications increase smoking cessation success and reduce withdrawal symptoms. The first-line pharmacotherapy agents include: bupropion SR, nicotine gum, nicotine inhaler, nicotine nasal spray, and the nicotine patch.
Population	Intensive intervention programs may be used with all tobacco users willing to participate in such efforts.

Intensive Strategy. Components of an intensive intervention.

efficacy of various treatments, assessments, and strategies for their implementation. This chapter addresses such questions as: Does the professional discipline of the clinician make a difference in the efficacy of the intervention? Are different types of providers effective in delivering interventions? Are minimal interventions, such as clinician advice to quit smoking, effective or are more intensive interventions required? Does the duration of an intervention in number of treatment sessions or in total face-to-face contact time substantially influence efficacy? Are pharmacologic interventions effective, and if so, which ones? Which counseling strategies are particularly helpful? In short, which treatments or assessments are efficacious and how should they be used?

The panel examined the relation between outcomes and 10 major assessment or treatment characteristics or strategies. These 10 characteristic types, and the categories within each, are listed in Table 6. Type of outcome varied across the different characteristics being analyzed. In most analyses, long-term abstinence was the outcome measure, in others it was the rate of smoker identification. The analyses reported in this chapter almost exclusively addressed treatments for cigarette smoking, as opposed to the use of other forms of tobacco, as the small number of studies on the use of noncigarette tobacco products precluded their separate analysis. Finally, the panel attempted to analyze treatment and assessment strategies that constitute distinct approaches that exist in current clinical practice.

The panel chose categories within each characteristic according to three major criteria. First, some categories reflected generally accepted dimensions or taxonomies. An example of this is the categorical nature of the clinician types (physician, psychologist, nurse, and so on). Second, information on the category had to be available in the published literature. Many questions of theoretical interest had to be abandoned simply because the requisite information was not available. Third, the category had to occur with sufficient frequency to permit meaningful statistical analysis. For example, the cut-points of some continuous variables (eg, total amount of contact time) were determined so there was a sufficient number of studies within each analytical category to permit meaningful analysis.

In ideal circumstances, the panel could evaluate each characteristic by consulting randomized controlled trials relevant to the specific categories in question. Unfortunately, with the exception of pharmacologic interventions, very few or no randomized controlled trials are specifically designed to address the effects of the various cate-

Action	**Strategies for Implementation**	
Implement an office-wide system that ensures that, for EVERY patient at EVERY clinic visit, tobacco-use status is queried and documented.	Office system change:	Expanding the Vital Signs to include tobacco use or implement an alternative universal identification system.
	Responsible staff:	Nurse, medical assistant, receptionist, or other individual already responsible for measuring the vital signs. These staff must be instructed regarding the importance of this activity and serve as non-smoking role models.
	Frequency of utilization:	Every visit for every patient regardless of the reason for the visit. (Repeated assessment is *not* necessary in the case of the adult who has never used tobacco or not used tobacco for many years, and for whom this information is clearly documented in the medical record.)
	System implementation steps:	Prepare progress notepaper or computer records to include tobacco use along with the traditional vital signs for every patient visit. A vital sign stamp also can be used. Alternatives to the vital sign stamp are to place tobacco-use status stickers on all patient charts or to indicate smoking status using computer reminder systems. VITAL SIGNS Blood Pressure: ____________________ Pulse: ______ Weight: ________ Temperature: ______________________ Respiratory Rate: ___________________ Tobacco Use: Current Former Never *(circle one)*

Systems Strategy 1. Implement a tobacco-user identification system in every clinic.

Action	**Strategies for Implementation**
Health care systems should ensure that clinicians have sufficient training to treat tobacco dependence, clinicians and patients have cessation resources, and clinicians are given feedback about their tobacco dependence treatment practices.	*Educate*: On a regular basis, offer lectures/seminars/ in-services with continuing medical education and/or other credit for tobacco dependence treatment. *Provide resources*: Have patient self-help materials as well as bupropion SR and nicotine replacement "starter kits" readily available in every examination room. Report: Include the provision of tobacco dependence treatment on "report cards" for managed care organizations and other insurers (eg, National Committee for Quality Assurance's Health Plan Employer Data and Information Set [NCQA HEDIS]). *Provide feedback*: Drawing on data from chart audits, electronic medical records, and computerized patient databases, evaluate the degree to which clinicians are identifying, documenting, and treating patients who use tobacco, and provide feedback to clinicians about their performance.

Systems Strategy 2. Provide education, resources, and feedback to promote provider intervention.

gories related to these treatment or assessment characteristics. Moreover, treatment characteristics are frequently confounded with one another. For example, comparisons among clinicians are almost always confounded with the type of counseling and behavioral therapy, format, and intensity of the interventions. Psychologists tend to deliver relatively intensive interventions, whereas physicians tend to deliver brief advice to individuals. Therefore, direct, unconfounded comparisons of categories within a particular characteristic were often impossible. These character-

Action	Strategies for Implementation
Clinical sites should communicate to all staff the importance of intervening with tobacco users and should designate a staff person (eg, nurse, medical assistant, or other clinician) to coordinate tobacco dependence treatments. Nonphysician personnel may serve as effective but lower cost providers of tobacco dependence interventions.	*Designate* a tobacco dependence treatment coordinator for every clinical site. *Delineate* the responsibilities of the tobacco dependence treatment coordinator, including instructing patients on the effective use of treatments (eg, pharmacotherapy, telephone calls to and from prospective quitters, and scheduled followup visits, especially in the immediate post-quit period). *Communicate* to each staff member (eg, nurse, physician, medical assistant, pharmacist, or other clinician) his or her responsibilities in the delivery of tobacco cessation services. Incorporate a discussion of these staff responsibilities into training of new and temporary staff.

Systems Strategy 3. Dedicate staff to provide tobacco dependence treatment and assess the delivery of this treatment in staff performance evaluations.

Action	Strategies for Implementation
Provide tobacco dependence treatment to all tobacco users admitted to a hospital.	*Implement* a system to identify and document the tobacco-use status of all hospitalized patients. *Identify* a clinician(s) to deliver tobacco dependence inpatient consultation services for every hospital. *Offer* tobacco dependence treatment to all hospitalized patients who use tobacco. *Reimburse* providers for tobacco dependence inpatient consultation services. *Expand* hospital formularies to include FDA-approved tobacco dependence pharmacotherapies. *Ensure* compliance with Joint Commission on Accreditation of Health Care Organizations regulations mandating that all sections of the hospital be entirely smoke-free. *Educate* hospital staff that first-line medications may be used to reduce nicotine withdrawal symptoms, even if the patient is not intending to quit.

Systems Strategy 4. Promote hospital policies that support and provide inpatient tobacco dependence services.

Action	Strategies for Implementation
Provide all insurance subscribers, including members of managed care organizations, with coverage for effective tobacco dependence treatments, including pharmacotherapy and counseling.	*Cover*: Include effective tobacco dependence treatments (both counseling and pharmacotherapy) as part of the basic benefits package for all health insurance packages. *Educate*: Inform subscribers, including members of managed care organizations, of the availability of covered tobacco dependence treatments (both counseling and pharmacotherapy) and encourage patients to use these services.

Systems Strategy 5. Include tobacco dependence treatments (both counseling and pharmacotherapy) identified as effective in this guideline as paid or covered services for all subscribers or members of health insurance packages.

istics were nevertheless analyzed because of their clinical importance, and because it was possible to reduce confounding by careful selection of studies and by statistical control of some confounding factors.

Additional topics, which did not lend themselves to analysis because of a lack of published long-term abstinence data, yet were important and clinically relevant, were nevertheless considered by the panel through a review of the existing literature. The strength of evidence associated with these recommendations clearly indicate that they are not based on meta-analyzed randomized controlled trials.

The present chapter addresses the 10 treatment and assessment characteristics outlined in Table 6 and is divided into three sections: (A) screening and assessment, (B) treatment structure and intensity, and (C) treatment elements. For each topic, background information, clinical recom-

Action	Strategies for Implementation
Reimburse fee-for-service clinicians and specialists for delivery of effective tobacco dependence treatments; include tobacco dependence treatments in the defined duties of salaried clinicians and those working in capitated environments.	*Include* tobacco dependence treatment as a reimbursable activity for fee-for-service providers. *Inform* fee-for-service clinicians and specialists that they will be reimbursed for using effective tobacco dependence treatments. *Include* tobacco dependence intervention in the job descriptions and performance evaluations of salaried clinicians and specialists.

Systems Strategy 6. Reimburse clinicians and specialists for delivery of effective tobacco dependence treatments and include these interventions among the defined duties of clinicians.

mendations, and the basis for those recommendations are provided. As described in Chapter 1, each recommendation was given a strength of evidence classification based on the criteria shown in Table 7. Finally, for some topics, recommendations for further research are provided.

A. Screening and Assessment.

Screen for Tobacco Use. Recommendation: All patients should be asked if they use tobacco and should have their tobacco-use status documented on a regular basis. Evidence has shown that this significantly increases rates of clinician intervention. (Strength of Evidence = A) Recommendation: Clinic screening systems such as expanding the vital signs to include tobacco-use status, or the use of other reminder systems such as chart stickers or computer prompts are essential for the consistent assessment, documentation, and intervention with tobacco use. (Strength of Evidence = B)

A thorough review of articles published since the end-date of the original guideline literature review (December 31, 1994), revealed no studies that met criteria for the screening meta-analyses. Therefore, the panel decided to rely on the meta-analyses from the original guideline to determine the impact of tobacco screening systems. Such screening systems were evaluated in terms of their impact on two outcomes: the rate of tobacco cessation intervention by clinicians, and the rate of cessation by patients who smoke.

Identifying Tobacco Users: Impact on Clinical Intervention. Nine studies met the selection criteria from the original guideline and were analyzed using a random-effects meta-analysis to assess the impact of screening systems on the rate of smoking cessation intervention by clinicians. The results of this meta-analysis are shown in Table 8. Implementing clinic systems designed to increase the assessment and documentation of tobacco use status markedly increases the rate at which clinicians intervene with their patients who smoke.

Identifying Tobacco Users: Impact on Tobacco Cessation. Three studies met the selection criteria from the original guideline and were analyzed using a random-effects meta-analysis to assess the impact of identifying smokers on actual rates of smoking cessation. The results of this meta-analysis are shown in Table 9. These results suggest that having a clinic system in place that identifies smokers results in higher rates of smoking cessation, although this finding was not statistically significant and was based on a small number of studies.

Brief Strategy A1 (see Chapter 3A) and Systems Strategy A1 (see Chapter 5) detail an approach for including tobacco-use status as a vital sign with systematic prompts and reminders. Although the data assessing this intervention were exclusively gathered from cigarette smokers, the panel felt that these results were generalizable to all tobacco users. This approach is designed to produce consistent assessment and documentation of tobacco use. Evidence from controlled trials shows that this approach increases the probability that tobacco use is consistently assessed and documented.[12,81–85]

Future Research. The following topic regarding screening for tobacco use requires additional research:

- Additional evidence regarding the impact of screening systems on tobacco abstinence rates.

Specialized Assessment. Recommendation: Once a tobacco user is identified and advised to quit, the clinician should assess the patient's willingness to quit at this time. (Strength of Evidence = C)

- **If the patient is willing to make a quit attempt at this time, interventions identified as effective in this guideline should be initiated. (see Chapter 3A and 4)**
- **If the patient is unwilling to quit at this time, a motivational intervention should be provided. (see Chapter 3B)**

Recommendation: Tobacco dependence treatment is effective and should be delivered even if specialized

Table 6. Treatment and Assessment Characteristics Analyzed for Their Relation to Abstinence Outcomes

Characteristics Analyzed	Categories of Those Characteristics
Screen for Tobacco Use	No screening system in place Screening system in place
Advice to Quit	No advice to quit Physician advice to quit
Intensity of Person-to-Person Clinical Contact	No person-to-person intervention Minimal counseling (longest session ≤ 3 min in duration) Low intensity counseling (longest session > 3 min and ≤ 10 min in duration) Higher intensity counseling (longest session > 10 min) Total amount of contact time Number of person-to-person treatment sessions
Type of Clinician	No clinician Self-help materials only Nonphysician health care clinician (eg, psychologist, counselor, social worker, nurse, dentist, graduate student, pharmacist) Physician Number of types of clinicians
Formats of Psychosocial Intervention	No contact Self-help/self-administered (eg, pamphlet, audiotape, videotape, mailed information, computer program) Individual counseling/contact Group counseling/contact Proactive telephone counseling/contact Number of types of formats
Self-Help Interventions	Number of self-help interventions Self-help interventions
Types of Counseling and Behavioral Therapies	No person-to-person intervention or minimal counseling General—problem-solving/coping skills/relapse prevention/stress management approach Negative affect/depression intervention Weight/diet/nutrition intervention Extra-treatment social support intervention Intra-treatment social support intervention Contingency contracting/instrumental contingencies Rapid smoking Other aversive smoking techniques Cigarette fading/smoking reduction prequit Acupuncture
Pharmacologic Interventions	Placebo pharmacotherapy Bupropion SR Clonidine Nicotine gum Nicotine inhaler Nicotine nasal spray Nicotine patch Nortriptyline
Combination Nicotine Replacement Therapy	One nicotine replacement therapy Two nicotine replacement therapies
Over-the-Counter Pharmacotherapy	Placebo over-the-counter nicotine patch therapy Over-the-counter nicotine patch therapy

Table 7. Summary of Strength of Evidence for Recommendations

Strength of Evidence Classification	Criteria
Strength of Evidence = A	Multiple well-designed randomized clinical trials, directly relevant to the recommendation, yielded a consistent pattern of findings.
Strength of Evidence = B	Some evidence from randomized clinical trials supported the recommendation, but the scientific support was not optimal. For instance, few randomized trials existed, the trials that did exist were somewhat inconsistent, or the trials were not directly relevant to the recommendation.
Strength of Evidence = C	Reserved for important clinical situations where the panel achieved consensus on the recommendation in the absence of relevant randomized controlled trials.

Table 8. Meta-Analysis: Impact of Having a Tobacco Use Status Identification System in Place on Rates of Clinician Intervention with Their Patients Who Smoke (n = 9 studies)

Screening System	Number of Arms	Estimated Odds Ratio (95% CI)	Estimated Intervention Rate (95% CI)
No screening system in place to identify smoking status (reference group)	9	1.0	38.5
Screening system in place to identify smoking status	9	3.1 (2.2–4.2)	65.6 (58.3–72.6)

CI = confidence interval

Table 9. Meta-Analysis: Impact of Having a Tobacco Use Status Identification System in Place on Abstinence Rates Among Patients Who Smoke (n = 3 studies)

Screening System	Number of Arms	Estimated Odds Ratio (95% CI)	Estimated Abstinence Rate (95% CI)
No screening system in place to identify smoking status (reference group)	3	1.0	3.1
Screening system in place to identify smoking status	3	2.0 (0.8–4.8)	6.4 (1.3–11.6)

CI = confidence interval

Table 10. Variables Associated with Higher or Lower Abstinence Rates

Variables Associated with Higher Abstinence Rates	Examples
High motivation	Tobacco user reports motivation to quit.
Ready to change	Tobacco user is ready to quit within a 1-month period.
Moderate to high self-efficacy	Tobacco user is confident in his or her ability to quit.
Supportive social network	A smoke-free workplace and home; friends who do not smoke in the quitter's presence.
Variables Associated with Lower Abstinence Rates	**Examples**
High nicotine dependence	Tobacco user has had severe withdrawal during previous quit attempts, smokes heavily (> 20 cigarettes/d), and/or has first cigarette of the day within 30 minutes after waking in the morning.
History of psychiatric comorbidity	Tobacco user has a history of depression, schizophrenia, alcoholism, or other chemical dependency.
High stress level	Stressful life circumstances and/or recent or anticipated major life changes (eg, divorce, job change, and marriage).

assessments are not used or available. (Strength of Evidence = A)

Every individual entering a health care setting should receive an assessment that determines his or her tobacco use status and interest in quitting. The patient should be asked, "Are you willing to make a quit attempt at this time?" Such an assessment (willing or unwilling) is a necessary first step in treatment. In addition, every patient should be assessed for physical or medical conditions that may affect the use of planned treatments (eg, pharmacotherapy).

The clinician also may want to perform specialized assessments of individual and environmental attributes that provide information for tailoring treatment and that predict quitting success. Specialized assessments refer to the use of formal instruments (eg, questionnaires, clinical interviews, or physiologic indices such as carbon monoxide, serum nicotine/cotinine levels, and/or pulmonary function) that may be associated with cessation outcome. Some of the variables targeted by specialized assessments that predict quitting success are listed in Table 10.

Several considerations should be kept in mind regarding the use of specialized assessments. First, there is little consistent evidence that a smoker's status on a specialized assessment is useful for treatment matching. The one exception is that persons highly nicotine dependent may benefit more from 4 mg as opposed to 2 mg nicotine gum (see Chapter 6C, Pharmacotherapy). More importantly, the panel found that, regardless of their standing on specialized assessments, all smokers have the potential to benefit from cessation interventions. Therefore, delivery of tobacco dependence treatments should not depend on the use of specialized assessments. Finally, tailored interventions based on specialized assessments (eg, stages of change) do not consistently produce higher long-term quit rates than do nontailored interventions of equal intensity. There do exist, however, some promising studies that suggest that individualizing self-help materials may be beneficial.[86,87] However, more studies are needed that contrast individualized self-help interventions with nonindividualized interventions of equivalent intensity. In addition, the panel recognizes that some effective interventions, such as general problem-solving (see Chapter 6C, Types of Counseling and Behavioral Therapies), entail treatment tailoring based on a systematic assessment of individual patient characteristics.

The existing evidence suggests that treatment can be effective despite the presence of risk factors for relapse (eg, severe previous withdrawal, depression, other smokers in the home), but abstinence rates in smokers with these characteristics tend to be lower than rates in those without these characteristics.[88–92]

Future Research. The following topic regarding specialized assessment requires additional research:

- Whether treatment adjustment based on specialized assessments can improve long-term abstinence rates.

B. Treatment Structure and Intensity

Advice To Quit Smoking. Recommendation: All physicians should strongly advise every patient who smokes to quit because evidence shows that physician advice to quit smoking increases abstinence rates. (Strength of Evidence = A)

Recommendation: All *clinicians* should strongly advise their patients who use tobacco to quit. Although studies have not independently addressed the impact of advice to quit by all types of nonphysician clinicians, it is reasonable to believe that such advice is effective in increasing their patients' long-term quit rates. (Strength of Evidence = B)

A thorough literature review identified no new studies since 1994 that examined the efficacy of advice to quit and that met the inclusion criteria for analysis. Therefore, the panel decided to rely on the analysis from the original guideline. Seven studies were included in the meta-analysis of the efficacy of physician advice to quit smoking. In the studies used in this analysis, the modal length of clinician intervention was 3 minutes or less. Two studies in this analysis used interventions lasting about 5 minutes. Results of the meta-analysis on physician advice are shown in Table 11. This analysis shows that brief physician advice significantly increases long-term smoking abstinence rates.

Table 11. Meta-Analysis: Efficacy of and Estimated Abstinence Rates for Advice to Quit by a Physician ($n = 7$ studies)

Advice	Number of Arms	Estimated Odds Ratio (95% CI)	Estimated Abstinence Rate (95% CI)
No advice to quit (reference group)	9	1.0	7.9
Physician advice to quit	10	1.3 (1.1–1.6)	10.2 (8.5–12.0)

CI = confidence interval

Physician advice only was examined in the Table 11 meta-analysis because there were too few studies to examine advice delivered by any other types of clinicians. The analysis for total amount of contact time (see Table 13 provided in the following subsection) indicates that minimal counseling (advice) delivered by a variety of clinician types increase long-term abstinence rates. Also, it should be noted that studies have shown that dentists and dental hygienists can be effective in assessing and advising patients who use smokeless/spit tobacco to quit (see Chapter 8). Given the large number of smokers who visit a clinician each year, the potential public health impact of universal advice to quit is substantial.

Future Research. The following topics regarding advice to quit require additional research:

- The efficacy of advice to quit smoking given by specific nonphysician clinicians such as nurses, psychologists, pharmacists, dentists, and dental hygienists.
- The cumulative efficacy of combined advice from physicians and nonphysician providers.

Intensity of Clinical Interventions. Recommendation: Minimal interventions lasting less than 3 minutes increase overall tobacco abstinence rates. Every tobacco user should be offered at least a minimal intervention whether or not he or she is referred to an intensive intervention. (Strength of Evidence = A)

Recommendation: There is a strong dose-response relation between the session length of person-to-person contact and successful treatment outcomes. Intensive interventions are more effective than less intensive interventions and should be used whenever possible. (Strength of Evidence = A)

Table 12. Meta-Analysis: Efficacy of and Estimated Abstinence Rates for Various Intensity Levels of Person-to-Person Contact (n = 43 studies)

Level of Contact	Number of Arms	Estimated Odds Ratio (95% CI)	Estimated Abstinence Rate (95% CI)
No contact	30	1.0	10.9
Minimal counseling (< 3 min)	19	1.3 (1.01, 1.6)	13.4 (10.9, 16.1)
Low intensity counseling (3–10 min)	16	1.6 (1.2, 2.0)	16.0 (12.8, 19.2)
Higher intensity counseling (> 10 min)	55	2.3 (2.0, 2.7)	22.1 (19.4, 24.7)

CI = confidence interval

Recommendation: Person-to-person treatment delivered for four or more sessions appears especially effective in increasing abstinence rates. Therefore, if feasible, clinicians should strive to meet four or more times with individuals quitting tobacco use. (Strength of Evidence = A)

These recommendations are supported by three separate analyses, one involving session length, one involving total amount of contact time, and one involving the number of sessions.

Session Length. Forty-three studies met selection criteria for comparisons among various session lengths. Whenever possible, session length was categorized based on the maximum amount of time the clinician spent with a smoker addressing tobacco dependence in a single contact. Minimal counseling interventions were defined as 3 minutes or less, low intensity counseling was defined as greater than 3 minutes to 10 minutes, and higher intensity counseling interventions were defined as greater than 10 minutes. Interventions could involve multiple patient-clinician contacts with the session length determined for coding purposes as the length of time of the longest session. These levels of person-to-person contact were compared with a no-contact reference group involving study conditions where subjects received no person-to-person contact (eg, self-help-only conditions). There is a dose-response relation between session length and abstinence rates. As Table 12 shows, all three session lengths (minimal counseling, low intensity counseling, and higher intensity counseling) significantly increased abstinence rates over those produced by no-contact conditions. However, there was a clear trend for abstinence rates to increase across these session lengths, with higher intensity counseling producing abstinence rates that were significantly higher than the rates produced by minimal or low intensity counseling.

Table 13. Meta-Analysis: Efficacy of and Estimated Abstinence Rates for Total Amount of Contact Time (n = 35 studies)

Total Contact Time	Number of Arms	Estimated Odds Ratio (95% CI)	Estimated Abstinence Rate (95% CI)
No min	16	1.0	11.0
1–3 min	12	1.4 (1.1, 1.8)	14.4 (11.3, 17.5)
4–30 min	20	1.9 (1.5, 2.3)	18.8 (15.6, 22.0)
31–90 min	16	3.0 (2.3, 3.8)	26.5 (21.5, 31.4)
91–300 min	16	3.2 (2.3, 4.6)	28.4 (21.3, 35.5)
> 300 min	15	2.8 (2.0, 3.9)	25.5 (19.2, 31.7)

CI = confidence interval

Total Amount of Contact Time. Thirty-five studies met the selection criteria for the analysis assessing the impact of total contact time. The amount of contact time was calculated from the text as the total time accumulated (the number of sessions multiplied by the session length). When the exact time was not known for minimal and low intensity interventions, they were assigned median lengths of 2 and 6.5 minutes, respectively. The total amount of contact time was then categorized as no-contact, 1–3 minutes, 4–30 minutes, 31–90 minutes, 91–300 minutes, and greater than 300 minutes. There is a dose-response relation between total amount of contact time and abstinence rates. As Table 13 shows, any contact time significantly increased abstinence rates over those produced by no-contact. However, there was a clear trend for abstinence rates to increase across contact time, with 31–90 minutes producing abstinence rates that were significantly higher than the rates produced by 1–3 minutes of total contact time. There was no evidence that more than 90 minutes of contact time increases abstinence rates further.

Number of Sessions. Forty-five studies involving at least some person-to-person contact met selection criteria for the analysis addressing the impact of number of treatment sessions. The number of treatment sessions was categorized as zero or one session, two to three sessions, four to eight sessions, and greater than eight sessions. Zero or one session was used as the reference group. As shown in Table 14, multiple treatment sessions increase smoking abstinence rates over those produced by zero or one session. The evidence suggests a dose-response relation between number of sessions and treatment efficacy, with treatments lasting more than eight sessions significantly more effective than interventions lasting either zero to one or two to three sessions.

Future Research. The following topics regarding intensity of person-to-person contact require additional research:

Table 14. Meta-Analysis: Efficacy of and Estimated Abstinence Rates for Number of Person-to-Person Treatment Sessions (*n* = 45 studies)

Number of Sessions	Number of Arms	Estimated Odds Ratio (95% CI)	Estimated Abstinence Rate (95% CI)
0–1 session	43	1.0	12.4
2–3 sessions	17	1.4 (1.1, 1.7)	16.3 (13.7, 19.0)
4–8 sessions	23	1.9 (1.6, 2.2)	20.9 (18.1, 23.6)
> 8 sessions	51	2.3 (2.1, 3.0)	24.7 (21.0, 28.4)

CI = confidence interval

- The effects of treatment duration and spacing of sessions (ie, the number of days or weeks over which treatment is spread). For instance, does front-loading sessions (having the majority of the sessions during the first few weeks of a quit attempt), or spacing sessions throughout the quit attempt yield better long-term abstinence rates?
- Methods to increase the patient utilization and completion of intensive treatments.
- Efficacy of intensive inpatient treatment programs.

Type of Clinician. Recommendation: Treatment delivered by a variety of clinician types increases abstinence rates. Therefore, all clinicians should provide smoking cessation interventions. (Strength of Evidence = A)

Recommendation: Treatments delivered by multiple types of clinicians are more effective than interventions delivered by a single type of clinician. Therefore, if feasible, the delivery of interventions by more than one type of clinician is encouraged. (Strength of Evidence = C)

Clinician Types. Twenty-nine studies met selection criteria for the analysis examining the effectiveness of various types of clinicians providing smoking cessation interventions. These analyses compared the efficacy of interventions delivered by specific types of clinicians with interventions where there were no clinicians (eg, where there was no intervention or the intervention consisted of self-help materials only). Smoking cessation interventions delivered by any single type of health care provider, such as a physician or nonphysician clinician (eg, psychologist, nurse, dentist, or counselor), or by multiple clinicians, increase abstinence rates relative to interventions where there is no clinician (eg, self-help interventions). None of the studies in these analyses involved pharmacotherapy, but they did involve psychosocial intervention of varying intensities. Results are shown in Table 15. Results are consistent across diverse clinician groups, with no clear advantage to any single clinician type.

Table 15. Meta-Analysis: Efficacy of and Estimated Abstinence Rates for Interventions Delivered by Various Types of Clinicians (*n* = 29 studies)

Type of Clinician	Number of Arms	Estimated Odds Ratio (95% CI)	Estimated Abstinence Rate (95% CI)
No clinician	16	1.0	10.2
Self-help	47	1.1 (0.9, 1.3)	10.9 (9.1, 12.7)
Nonphysician clinician	39	1.7 (1.3, 2.1)	15.8 (12.8, 18.8)
Physician clinician	11	2.2 (1.5, 3.2)	19.9 (13.7, 26.2)

CI = confidence interval

Table 16. Meta-Analysis: Efficacy of and Estimated Abstinence Rates for Interventions Delivered by Various Numbers of Clinician Types (*n* = 37 studies)

Number of Clinician Types	Number of Arms	Estimated Odds Ratio (95% CI)	Estimated Abstinence Rate (95% CI)
No clinician	30	1.0	10.8
One clinician type	50	1.8 (1.5, 2.2)	18.3 (15.4, 21.1)
Two clinician types	16	2.5 (1.9, 3.4)	23.6 (18.4, 28.7)
Three or more clinician types	7	2.4 (2.1, 2.9)	23.0 (20.0, 25.9)

CI = confidence interval

Number of Clinician Types. Thirty-seven studies met selection criteria for the analysis examining the effectiveness of multiple clinicians used in smoking cessation interventions. Please note that “multiple clinicians” refers to the number of different types of clinicians, not the number of total clinicians regardless of type. The latter information was rarely available from the study reports. Smoking cessation interventions delivered by multiple types of clinicians increase abstinence rates relative to those produced by interventions where there is no clinician. Results are shown in Table 16. The data displayed in Table 16 also show a nonsignificant trend for multiple types of clinicians to be more efficacious than a single clinician type. This suggests that a variety of clinicians, including physician clinicians, and nonphysician clinicians, such as nurses, dentists, dental hygienists, psychologists, pharmacists, and health educators, can play an important role in promoting smoking cessation.

Future Research. The following topics regarding type of clinician require additional research:

- The effectiveness of specific types of clinicians, such as nurses, physician assistants, pharmacists, social workers, etc.

Table 17. Meta-Analysis: Efficacy of and Estimated Abstinence Rates for Various Types of Format ($n = 58$ studies)

Format	Number of Arms	Estimated Odds Ratio (95% CI)	Estimated Abstinence Rate (95% CI)
No format	20	1.0	10.8
Self-help	93	1.2 (1.02, 1.3)	12.3 (10.9, 13.6)
Proactive telephone counseling	26	1.2 (1.1, 1.4)	13.1 (11.4, 14.8)
Group counseling	52	1.3 (1.1, 1.6)	13.9 (11.6, 16.1)
Individual counseling	67	1.7 (1.4, 2.0)	16.8 (14.7, 19.1)

CI = confidence interval

- The relative effectiveness of various numbers and types of clinicians, with the intensity of the intervention held constant.
- Strategies to integrate tobacco dependence treatment across diverse disciplines and settings.

Formats of Psychosocial Treatments. Recommendation: Proactive telephone counseling, and group and individual counseling formats are effective and should be used in smoking cessation interventions. (Strength of Evidence = A)

Recommendation: Smoking cessation interventions that are delivered in multiple formats increase abstinence rates and should be encouraged. (Strength of Evidence = A)

Format Types. Fifty-eight studies met selection criteria and were included in the analysis comparing different types of formats. Smoking cessation interventions delivered by means of proactive telephone counseling/contact, individual counseling, and group counseling/contact all increase abstinence rates relative to no intervention.

This format meta-analysis also evaluated the efficacy of self-help interventions (eg, pamphlets/booklets/mailings/manuals, videotapes, audiotapes, referrals to 12-step programs, mass media community level interventions, reactive telephone hotlines/help lines, computer programs/Internet, and lists of community programs). Interventions delivered by means of widely varied self-help materials (whether as stand-alone treatments or as adjuvants) appear to increase abstinence rates relative to no intervention in this particular analysis. However, the effect of self-help is weak and inconsistent across analyses conducted for this guideline. The impact of self-help is certainly smaller and less certain than that of proactive telephone, individual, or group counseling. Results of this analysis are shown in Table 17.

Table 18. Meta-Analysis: Efficacy of and Estimated Abstinence Rates for Number of Formats ($n = 54$ studies)

Number of Formats*	Number of Arms	Estimated Odds Ratio (95% CI)	Estimated Abstinence Rate (95% CI)
No format	20	1.0	10.8
One format	51	1.5 (1.2, 1.8)	15.1 (12.8, 17.4)
Two formats	55	1.9 (1.6, 2.2)	18.5 (15.8, 21.1)
Three or four formats	19	2.5 (2.1, 3.0)	23.2 (19.9, 26.6)

*Formats included self-help, proactive telephone counseling, group, or individual counseling.
CI = confidence interval

Number of Formats. Fifty-four studies met selection criteria and were included in the analysis comparing the number of format types used for smoking cessation interventions. The self-help treatments included in this analysis occurred either by themselves or as adjuvants to other treatments. Smoking cessation interventions that used more than two format types were more effective than interventions that used a single format type. Results of this analysis are shown in Table 18.

Self-help: focused analyses. Because the format analysis revealed self-help to be of marginal efficacy, another analysis was undertaken to provide additional, focused information on self-help. Studies were accepted for this analysis if the presence of self-help materials constituted the sole difference in treatment arms. In the main format analysis, some treatment arms differed on factors other than self-help per se (eg, intensity of adjuvant counseling). The treatments that accompanied self-help material in the focused analysis ranged from no advice or counseling to intensive counseling. The results of this analysis were comparable to those in the larger format analysis (ie, self-help was of marginal efficacy).

Twenty-one studies met selection criteria to evaluate the efficacy of providing multiple types of self-help interventions (eg, pamphlets, videotapes, audiotapes, and reactive hotlines/help lines). The results provide little evidence that the provision of multiple types of self-help, when offered without any person-to-person intervention, significantly enhances treatment outcomes (Table 19).

There are two limitations to interpreting these results. First, self-help materials vary greatly in nature and intensity. It is possible that some sub-types of self-help are, in fact, efficacious (eg, those that are individualized). Second, a large number of smokers report that they quit on their own without clinical support or contact.[93] The extent to which use of self-help materials aids self-quitters was not addressed in guideline analyses.

The previous analyses failed to show a consistent, beneficial effect because of self-help. Two final meta-analyses addressed the impact of self-help brochures per se. In one

Table 19. Meta-Analysis: Efficacy of and Estimated Abstinence Rates for Number of Types of Self-Help (n = 21 studies)

Factor	Number of Arms	Estimated Odds Ratio (95% CI)	Estimated Abstinence Rate (95% CI)
No self-help	17	1.0	14.3
One type of self-help	27	1.0 (0.9, 1.1)	14.4 (12.9, 15.9)
Two or more types	10	1.1 (0.9, 1.5)	15.7 (12.3, 19.2)

CI = confidence interval

analysis, brochures were used as the only intervention. In the other analysis, self-help brochures were used as adjuvants to counseling. In neither analysis did self-help significantly boost abstinence rates.

Future Research. The following topics regarding formats require additional research:

- Identify which combinations of formats are effective.
- The efficacy of innovative approaches to self-help such as individualized computerized interventions.[87,94,95].
- The efficacy of reactive telephone hotlines/help lines.[60,96–99]
- The relative efficacy of different types of self-help interventions.
- The efficacy of self-help materials as adjuvant treatments. Do they add significantly to the effectiveness of other proven tobacco dependence treatments such as individual counseling, group counseling, proactive telephone counseling, and pharmacotherapy?

Follow-up Assessment and Procedures. Recommendation: All patients who receive a tobacco dependence intervention should be assessed for abstinence at the completion of treatment and during subsequent clinic contacts.[1] Abstinent patients should receive relapse prevention treatment (see Chapter 3C, For the Patient Who Has Quit).[2] Patients who have relapsed should be assessed to determine whether they are willing to make another quit attempt. (Strength of Evidence = C):

- **If the patient is willing to make another quit attempt, provide or arrange additional treatment (see Chapter 3A, For the Patient Willing To Quit).**
- **If the patient is not willing to try to quit, provide an intervention to promote motivation to quit (see Chapter 3B, For the Patient Unwilling To Quit).**

All patients should be assessed with respect to their smoking status during all follow-up tobacco dependence contacts. In particular, assessments within the first week after quitting also should be encouraged.[100] Abstinent patients should receive relapse prevention treatment (see Chapter 3C, Brief Strategy C1 and Brief Strategy C2) including reinforcement for their decision to quit, congratulations on their success at quitting, and encouragement to remain abstinent.[62] Clinicians also should inquire about current and future threats to abstinence and provide appropriate suggestions for coping with these threats.

Patients who have relapsed should again be assessed for their willingness to quit. Patients who are currently motivated to make another quit attempt should be provided with a tobacco dependence intervention (see Chapter 3A, For the Patient Willing To Quit). Clinicians may wish to increase the intensity of psychosocial treatment at this time or refer the patient to a tobacco dependence specialist/program for a more intensive treatment if the patient is willing. In addition, pharmacotherapy should be again offered to the patient. If the previous cessation attempt included pharmacotherapy, the clinician should review whether the patient used these medications in an effective manner and determine whether the medication was helpful. Based on this assessment, recommend retreatment with the same pharmacotherapy, another pharmacotherapy, or combination nicotine replacement therapies (see Tables 33–39).

Patients who are unwilling to quit at the current time should receive a brief intervention designed to promote the motivation to quit (see Chapter 3B, Brief Strategy B. Enhancing motivation to quit tobacco—the "5 R's").

Future Research. The following topics regarding follow-up assessment require additional research:

- The optimal timing and types of relapse prevention interventions.
- The efficacy of various formats for relapse prevention treatments. For instance, are telephone contacts effective in reducing the likelihood of relapse after a minimal intervention?

C. Treatment Elements

Types of Counseling and Behavioral Therapies. Recommendation: Three types of counseling and behavioral therapies result in higher abstinence rates: (1) providing smokers with practical counseling (problem-solving skills/skills training), (2) providing social support as part of treatment, and (3) helping smokers obtain social support outside of treatment. These types of counseling and behavioral therapies should be included in smoking cessation interventions. (Strength of Evidence = B)

Recommendation: Aversive smoking interventions (rapid smoking, rapid puffing, other aversive smoking techniques) increase abstinence rates and may be used with smokers who desire such treatment or who have been unsuccessful using other interventions. (Strength of Evidence = B)

Sixty-two studies met selection criteria for analyses examining the effectiveness of interventions using various

Table 20. Meta-Analysis: Efficacy of and Estimated Abstinence Rates for Various Types of Counseling and Behavioral Therapies ($n = 62$ studies)

Type of Counseling and Behavioral Therapy	Number of Arms	Estimated Odds Ratio (95% CI)	Estimated Abstinence Rate (95% CI)
No counseling behavioral therapy	35	1.0	11.2
Relaxation/breathing	31	1.0 (0.7, 1.3)	10.8 (7.9, 13.8)
Contingency contracting	22	1.0 (0.7, 1.4)	11.2 (7.8, 14.6)
Weight/diet	19	1.0 (0.8, 1.3)	11.2 (8.5, 14.0)
Cigarette fading	25	1.1 (0.8, 1.5)	11.8 (8.4, 15.3)
Negative affect	8	1.2 (0.8, 1.9)	13.6 (8.7, 18.5)
Intra-treatment social support	50	1.3 (1.1, 1.6)	14.4 (12.3, 16.5)
Extra-treatment social support	19	1.5 (1.1, 2.1)	16.2 (11.8, 20.6)
General—problem-solving	104	1.5 (1.3, 1.8)	16.2 (14.0, 18.5)
Other aversive smoking	19	1.7 (1.04, 2.8)	17.7 (11.2, 24.9)
Rapid smoking	19	2.0 (1.1, 3.5)	19.9 (11.2, 29.0)

CI = confidence interval

Table 21. Common Elements of Practical Counseling (Problem-Solving/Skills Training)

Practical Counseling (Problem-Solving/Skills Training) Treatment Component	Examples
Recognize danger situations: Identify events, internal states, or activities that increase the risk of smoking or relapse	• Negative affect • Being around other smokers • Drinking alcohol • Experiencing urges • Being under time pressure
Develop coping skills: Identify and practice coping or problem-solving skills. Typically, these skills are intended to cope with danger situations.	• Learning to anticipate and avoid temptation • Learning cognitive strategies that will reduce negative moods • Accomplishing lifestyle changes that reduce stress, improve quality of life, or produce pleasure • Learning cognitive and behavioral activities to cope with smoking urges (eg, distracting attention)
Provide basic information about smoking and successful quitting.	• The fact that any smoking (even a single puff) increases the likelihood of a full relapse • Withdrawal typically peaks within 1–3 weeks after quitting • Withdrawal symptoms include negative mood, urges to smoke, and difficulty concentrating • The addictive nature of smoking

types of counseling and behavioral therapies. The results, shown in Table 20 reveal that four specific types of counseling and behavioral therapy categories yield statistically significant increases in abstinence rates relative to no-contact (eg, untreated control conditions). These categories are: (1) providing practical counseling such as problem-solving/skills training/relapse prevention/stress management, (2) providing support during a smoker's direct contact with a clinician (intra-treatment social support), (3) intervening to increase social support in the smoker's environment (extra-treatment social support), and (4) using aversive smoking procedures (rapid smoking, rapid puffing, other smoking exposure). A separate analysis was conducted eliminating studies that included the use of United States Food and Drug Administration (FDA)-approved pharmacotherapies. The results of this analysis were substantially similar to the main analysis.

The strength of evidence for the recommendations regarding types of counseling and behavioral therapy categories did not warrant an "A" rating for several reasons. First, smoking cessation interventions rarely used a particular type of counseling or behavioral therapy in isolation. Second, various types of counseling and behavioral therapies tended to be correlated with other treatment characteristics. For instance, some types of counseling and behavioral therapies were more likely to be delivered using a greater number of sessions across longer time periods. Third, it must be noted that all of these types of counseling and behavioral therapies were compared with no-contact/control conditions. Therefore, the control conditions in this meta-analysis did not control for nonspecific or placebo effects of treatment. This further restricted the ability to attribute efficacy to particular types of counseling and behavioral therapies per se. Fourth, the studies used in this analysis often tailored the types of counseling and behavioral therapies to the needs of special populations being studied, thereby affecting the generalizability of the study results. Fifth, there was considerable heterogeneity within each type of counseling and behavioral therapy.

In the types of counseling and behavioral therapies meta-analysis, six studies examined the effect of dieting and physical activity interventions on smoking cessation. Although dieting and physical activity did not significantly increase abstinence rates based on that analysis, a single recent study published after the date for inclusion in meta-analysis found that vigorous exercise did boost quit rates.[101]

The treatments targeting negative affect were administered both to general populations as well as to special populations (eg, smokers with a history of depression).[102,103] It is possible that different results would have been found if the study arms were restricted to smokers at risk for negative affect.

Tables 21, 22, and 23 outline elements of practical counseling (problem-solving/skills training), intra-treatment social support, and extra-treatment social support respec-

Table 22. Common Elements of Intra-Treatment Supportive Interventions

Supportive Treatment Component	Examples
Encourage the patient in the quit attempt	• Note that effective tobacco dependence treatments are now available. • Note that half of all people who have ever smoked have now quit. • Communicate belief in patient's ability to quit.
Communicate caring and concern	• Ask how patient feels about quitting. • Directly express concern and willingness to help. • Be open to the patient's expression of fears of quitting, difficulties experienced, and ambivalent feelings.
Encourage the patient to talk about the quitting process	Ask about: • Reasons the patient wants to quit. • Concerns or worries about quitting. • Success the patient has achieved. • Difficulties encountered while quitting.

tively. These tables are designed to help clinicians using these counseling and behavioral therapies. It must be noted, however, that these treatment labels are nonspecific and include heterogeneous treatment elements.

Another type of behavioral therapy associated with superior outcomes is aversive smoking. This involves sessions of guided smoking where the patient smokes intensively, often to the point of discomfort, malaise, nausea, and/or vomiting. Some aversive smoking techniques, such as rapid smoking, may constitute a health risk and should be conducted only with appropriate medical screening and supervision. Aversive smoking interventions are infrequently used today.

Table 23. Common Elements of Extra-Treatment Supportive Interventions

Supportive Treatment Component	Examples
Train patient in support solicitation skills	• Show videotapes that model support skills. • Practice requesting social support from family, friends, and coworkers. • Aid patient in establishing a smoke-free home.
Prompt support seeking	• Help patient identify supportive others. • Call the patient to remind him or her to seek support. • Inform patients of community resources such as hotlines and helplines.
Clinician arranges outside support.	• Mail letters to supportive others. • Call supportive others. • Invite others to cessation sessions. • Assign patients to be "buddies" for one another.

Table 24. Meta-Analysis: Efficacy of and Estimated Abstinence Rates for Acupuncture ($n = 5$ studies)

Treatment	Number of Arms	Estimated Odds Ratio (95% CI)	Estimated Abstinence Rate (95% CI)
Placebo	7	1.0	8.3
Acupuncture	8	1.1 (0.7, 1.6)	8.9 (5.5, 12.3)

CI = confidence interval

Acupuncture. A separate meta-analysis was conducted for acupuncture This analysis was conducted to achieve a sensitive test on the small body of studies that use this technique. Evidence, as shown in Table 24, did not support the efficacy of acupuncture as a smoking cessation treatment. The acupuncture meta-analysis comparing "active" acupuncture with "control" acupuncture revealed no difference in efficacy between the two types of procedures. These results suggest that any effect of acupuncture might be produced by factors such as positive expectations about the procedure.

Hypnosis. The original guideline did not conduct a separate meta-analysis on hypnosis because few studies met inclusion criteria, and those that did used very heterogeneous hypnotic procedures. There was no common or standard intervention technique to analyze. Literature screening for the updated guideline revealed no new published studies on the treatment of tobacco dependence by hypnosis that met the inclusion criteria; therefore, this topic did not warrant re-examination. Moreover, an independent review of hypnotherapy trials by the Cochrane Group found insufficient evidence to support hypnosis as a treatment for smoking cessation.[104]

Other Interventions. There were insufficient studies to address the efficacy of other types of counseling and behavioral therapies such as physiologic feedback and restricted environmental stimulation therapy.

Future Research. The following topics regarding types of counseling and behavioral therapies require additional research:

- Motivational interventions, cigarette fading, hypnosis, physiologic feedback of smoking impacts, 12-step models, and restricted environmental stimulation therapy.
- Mechanisms through which counseling interventions exert their effects.

- Efficacy of specific counseling interventions among various patient populations (eg, those with cancers, COPD, and atherosclerosis).

Alternative Treatment Models for the Treatment of Tobacco Dependence—Stepped Care and Individual Tailoring. The panel concluded that there is not enough evidence to propose a recommendation regarding: (1) a stepped-care model for delivery of tobacco dependence treatment, and (2) individually tailored interventions (eg, using the transtheoretical model). Both of these intervention strategies hold promise, and enjoy some empirical support. Tailored self-help approaches especially enjoy some support.[105–107] Unfortunately, there is insufficient data to indicate that either approach yields a significant incremental impact on long-term abstinence rates over the impact of nontailored counseling approaches of similar intensity. As a result, there needs to be additional research to test the efficacy of these strategies. Some of the needed research includes:

- Whether the use of stepped-care intervention strategies improves long-term abstinence rates.
- Whether the use of treatment matching strategies improves long-term abstinence rates.
- Whether the use of tailored intervention strategies improves long-term abstinence rates.
- Whether targeted reductions in the number of cigarettes smoked per day increases long-term rates of abstinence.[108]
- Whether staged-based treatments developed in keeping with the transtheoretical model significantly improve long-term abstinence rates relative to comparably intense, alternative counseling strategies (eg, where patients receive only a standard motivational treatment or cessation treatment depending on their willingness to quit).

Pharmacotherapy. Recommendation: All patients attempting to quit should be encouraged to use effective pharmacotherapies for smoking cessation except in the presence of special circumstances. (Strength of Evidence = A)

Recommendation: Long-term smoking cessation pharmacotherapy should be considered as a strategy to reduce the likelihood of relapse (Strength of Evidence = C)

As with other chronic diseases, the most effective treatment of tobacco dependence requires the use of multiple modalities. Pharmacotherapy is a vital element of a multicomponent approach. The clinician should encourage all patients initiating a quit attempt to use one or a combination of efficacious pharmacotherapies, although pharmacotherapy use requires special consideration with some patient groups (eg, those with medical contraindications, those smoking fewer than 10 cigarettes a day, pregnant/breast-feeding women, and adolescent smokers). The guideline panel identified five first-line medications (bupropion SR, nicotine gum, nicotine inhaler, nicotine nasal spray, and the nicotine patch) and two second-line medications (clonidine and nortriptyline) for smoking cessation Each has been documented to significantly increase rates of long-term smoking abstinence No other pharmacotherapeutic treatments were supported by a consistent body of scientific evidence.

The pharmacotherapy meta-analyses were designed to compare particular pharmacotherapies with the placebo controls in each study. Because of substantial differences across the studies evaluating the different types of pharmacotherapies, it is inappropriate to compare the results for one medication with those for another in the tables that follow.

Pharmacotherapy meta-analyses included predominately studies with "self-selected" populations. In addition, in pharmacotherapy studies both experimental and control subjects typically received substantial counseling. Both of these factors tend to produce higher abstinence rates in reference or placebo subjects than are typically observed among self-quitters.

Recommendations Regarding Specific Pharmacotherapies: First-Line Medications

First-line pharmacotherapies have been found to be safe and effective for tobacco dependence treatment and have been approved by the FDA for this use. First-line medications have established empirical record of efficacy, and should be considered first as part of tobacco dependence treatment except in cases of contraindications.

The listing of the first-line medications is provided alphabetically. Meta-analyses did not contrast the relative efficacy of these medications.

Bupropion SR (Sustained Release Bupropion). Recommendation: Bupropion SR is an efficacious smoking cessation treatment that patients should be encouraged to use. (Strength of Evidence = A)

Two large multicenter studies met selection criteria and were included in the analysis comparing bupropion sustained release (SR) to placebo. Results of this analysis are shown in Table 25. As can be seen from this analysis, the use of bupropion SR approximately doubles long-term abstinence rates, compared to a placebo.

Bupropion SR is the first non-nicotine medication shown to be effective for smoking cessation and approved by the FDA for that use. Its mechanism of action is presumed to be mediated by its capacity to block neural re-uptake of dopamine and/or norepinephrine. It is contraindicated in patients with a seizure disorder, a current or prior diagnosis of bulimia or anorexia nervosa, use of a monoamine

Table 25. Meta-Analysis: Efficacy of and Estimated Abstinence Rates for Bupropion SR ($n = 2$ studies)

Pharmacotherapy	Number of Arms	Estimated Odds Ratio (95% CI)	Estimated Abstinence Rate (95% CI)
Placebo	2	1.0	17.3
Bupropion SR	4	2.1 (1.5, 3.0)	30.5 (23.2, 37.8)

CI = confidence interval

Table 26. Meta-Analysis: Efficacy of and Estimated Abstinence Rates for 2 mg Nicotine Gum ($n = 13$ studies)

Pharmacotherapy	Number of Arms	Estimated Odds Ratio (95% CI)	Estimated Abstinence Rate (95% CI)
Placebo	16	1.0	17.1
Nicotine gum	18	1.5 (1.3, 1.8)	23.7 (20.6, 26.7)

CI = confidence interval

Table 27. Meta-Analysis: Efficacy of and Estimated Abstinence Rates for Nicotine Inhaler ($n = 4$ studies)

Pharmacotherapy	Number of Arms	Estimated Odds Ratio (95% CI)	Estimated Abstinence Rate (95% CI)
Placebo	4	1.0	10.5
Nicotine inhaler	4	2.5 (1.7, 3.6)	22.8 (16.4, 29.2)

CI = confidence interval

Table 28. Meta-Analysis: Efficacy of and Estimated Abstinence Rates for Nicotine Nasal Spray ($n = 3$ studies)

Pharmacotherapy	Number of Arms	Estimated Odds Ratio (95% CI)	Estimated Abstinence Rate (95% CI)
Placebo	3	1.0	13.9
Nicotine nasal spray	3	2.7 (1.8, 4.1)	30.5 (21.8, 39.2)

CI = confidence interval

oxidase inhibitor within the previous 14 days, or in patients on another medication that contains bupropion. Bupropion SR can be used in combination with nicotine replacement therapies. Bupropion SR is available exclusively as a prescription medication both with an indication for smoking cessation (Zyban) and an indication for depression (Wellbutrin). Suggestions regarding the clinical use of bupropion SR are provided in Table 33.

Nicotine Gum. Recommendation: Nicotine gum is an efficacious smoking cessation treatment that patients should be encouraged to use. (Strength of Evidence = A)

Recommendation: Clinicians should offer 4 mg rather than 2 mg nicotine gum to highly dependent smokers. (Strength of Evidence = B)

Thirteen studies met selection criteria and were included in the analysis comparing nicotine gum to placebo. Results of this analysis are shown in Table 26. As can be seen by the estimated odds ratio from this analysis, 2 mg nicotine gum improves long-term abstinence rates by approximately 30–80%, compared with placebo. Furthermore, a close review of the literature suggests that the 4 mg gum is more efficacious than the 2 mg gum as an aid to smoking cessation in highly dependent smokers (see Table 10, Variables associated with higher or lower abstinence rates).[109,110]

Nicotine gum is currently available exclusively as an over-the-counter medication and is packaged with important instructions on correct usage, including chewing instructions. Suggestions regarding the clinical use of nicotine gum are provided in Table 34.

Nicotine Inhaler. Recommendation: The nicotine inhaler is an efficacious smoking cessation treatment that patients should be encouraged to use. (Strength of Evidence = A)

Four studies met selection criteria and were included in the analysis comparing the nicotine inhaler to placebo. Results of this analysis are shown in Table 27. As can be seen from this analysis, the nicotine inhaler more than doubles long-term abstinence rates, compared to a placebo inhaler.

The nicotine inhaler is available exclusively as a prescription medication. Suggestions regarding the clinical use of the nicotine inhaler are provided in Table 35.

Nicotine Nasal Spray. Recommendation: Nicotine nasal spray is an efficacious smoking cessation treatment that patients should be encouraged to use. (Strength of Evidence = A)

Three studies met selection criteria and were included in the analysis comparing nicotine nasal spray to placebo. Results of this analysis are shown in Table 28. As can be seen from this analysis, nicotine nasal spray more than doubles long-term abstinence rates, compared to a placebo spray.

Nicotine nasal spray is available exclusively as a prescription medication. Suggestions regarding the clinical use of the nicotine nasal spray are provided in Table 36.

Nicotine Patch. Recommendation: The nicotine patch is an efficacious smoking cessation treatment that patients should be encouraged to use. (Strength of Evidence = A)

Table 29. Meta-Analysis: Efficacy of and Estimated Abstinence Rates for the Nicotine Patch (n = 27 studies)

Pharmacotherapy	Number of Arms	Estimated Odds Ratio (95% CI)	Estimated Abstinence Rate (95% CI)
Placebo	28	1.0	10.0
Nicotine patch	32	1.9 (1.7, 2.2)	17.7 (16.0, 19.5)

CI = confidence interval

Twenty-seven studies met selection criteria and were included in the analysis comparing the nicotine patch to placebo. Results of this analysis are shown in Table 29. As can be seen from this analysis, the nicotine patch approximately doubles long-term abstinence rates over those produced by placebo interventions.

The nicotine patch is available both as an over-the-counter medication and as a prescription medication. Suggestions regarding clinical use of the nicotine patch are provided in Table 37.

Recommendations Regarding Specific Pharmacotherapies: Second-Line Medications

Second-line medications are pharmacotherapies for which there is evidence of efficacy for treating tobacco dependence, but they have a more limited role than first-line medications because: (1) the FDA has not approved them for a tobacco dependence treatment indication, and (2) there are more concerns about potential adverse effects than exist with first-line medications. Second-line treatments should be considered for use on a case-by-case basis after first-line treatments have been used or considered.

The listing of the second-line medications is provided alphabetically. Meta-analyses did not contrast the relative efficacy of these medications.

Clonidine. Recommendation: Clonidine is an efficacious smoking cessation treatment. It may be used under a physician's supervision as a second-line agent to treat tobacco dependence. (Strength of Evidence = A)

Five studies met selection criteria and were included in the analysis comparing clonidine to placebo. Results of this analysis are shown in Table 30. As can be seen from this analysis, the use of clonidine approximately doubles abstinence rates, compared to placebo. These studies varied the clonidine dose from 0.1 mg/d to 0.75 mg/d. The drug was delivered either transdermally or orally. It should be noted that abrupt discontinuation of clonidine can result in symptoms such as nervousness, agitation, headache, and tremor, accompanied or followed by a rapid rise in blood pressure and elevated catecholamine levels.

Table 30. Meta-Analysis: Efficacy of and Estimated Abstinence Rates for Clonidine (n = 5 studies)

Pharmacotherapy	Number of Arms	Estimated Odds Ratio (95% CI)	Estimated Abstinence Rate (95% CI)
Placebo	6	1.0	13.9
Clonidine	8	2.1 (1.4, 3.2)	25.6 (17.7, 33.6)

CI = confidence interval

Table 31. Meta-Analysis: Efficacy of and Estimated Abstinence Rates for Nortriptyline (n = 2 studies)

Pharmacotherapy	Number of Arms	Estimated Odds Ratio (95% CI)	Estimated Abstinence Rate (95% CI)
Placebo	3	1.0	11.7
Nortriptyline	3	3.2 (1.8, 5.7)	30.1 (18.1, 41.6)

CI = confidence interval

Clonidine is used primarily as an antihypertensive medication and has not been approved by the FDA as a smoking cessation medication. Therefore, clinicians need to be aware of the specific warnings regarding this medication, as well as its adverse effect profile.

Additionally, a specific dosing regimen for the use of clonidine has not been established. Because of the warnings associated with clonidine discontinuation, the variability in dosages used to test this medication, and a lack of FDA approval, the guideline panel chose to recommend clonidine as a second-line agent. As such, clonidine should be considered for smoking cessation under a physician's direction with patients unable to use first-line medications because of contraindications or with patients who were unable to quit using first-line medications. Suggestions regarding clinical use of clonidine are provided in Table 38.

Nortriptyline. Recommendation: Nortriptyline is an efficacious smoking cessation treatment. It may be used under a physician's supervision as a second-line agent to treat tobacco dependence. (Strength of Evidence = B)

Two studies met selection criteria and were included in the analysis comparing nortriptyline to placebo. Results of this analysis are shown in Table 31. As can be seen from this analysis, the use of nortriptyline increases abstinence rates, compared to a placebo.

Nortriptyline is used primarily as an antidepressant and has not been evaluated or approved by the FDA as a smoking cessation medication. Clinicians need to be aware of the specific warnings regarding this medication as well as its side effect profile. Because of the limited number of studies examining nortriptyline and the small sample sizes

Table 32. Meta-Analysis: Efficacy of and Estimated Abstinence Rates for Combination Nicotine Replacement Therapy ($n = 3$ studies)

Factor	Number of Arms	Odds Ratio	Estimated Abstinence Rate (95% CI)
One NRT	3	1.0	17.4
Two NRTs	3	1.9 (1.3, 2.6)	28.6 (21.7, 35.4)

NRT = nicotine replacement therapy
CI = confidence interval

within those studies, the guideline panel determined that the recommendation warranted a strength of evidence equal to B. Because of this strength of evidence, the side effect profile, and the lack of FDA approval for tobacco dependence treatment, nortriptyline is recommended as a second-line agent. As such, nortriptyline should be considered for smoking cessation under a physician's direction with patients unable to use first-line medications because of contraindications or with patients who were unable to quit using first-line medications. Suggestions regarding clinical use of nortriptyline are provided in Table 39.

Combination Nicotine Replacement Therapy. Recommendation: Combining the nicotine patch with a self-administered form of nicotine replacement therapy (either the nicotine gum or nicotine nasal spray) is more efficacious than a single form of nicotine replacement, and patients should be encouraged to use such combined treatments if they are unable to quit using a single type of first-line pharmacotherapy. (Strength of Evidence = B)

Three studies met selection criteria for the combination nicotine replacement therapy (NRT) meta-analysis. This analysis was intended to address the hypothesis that combination pharmacotherapy is more effective than monotherapy (the use of a single pharmacotherapy) if the combination therapy comprises two different types of pharmacotherapy. Specifically, the hypothesis holds that one type of pharmacotherapy should involve passive dosing that produces relatively steady levels of drug in the body, while the second type of pharmacotherapy should permit *ad libitum* dosing that allows the user to adjust dosing on an acute basis.[111,112] All three studies used the nicotine patch (15 mg) as one of the medications; in two studies the patch was supplemented with nicotine gum (2 mg),[113,114] and in the remaining study the patch was supplemented with nicotine nasal spray.[115] Comparison subjects receiving monotherapy were given the nicotine patch in two of these studies and nicotine gum in the third.

The results of the combination NRT meta-analysis are displayed in Table 32. The results show that the combination NRT treatment produced higher long-term abstinence rates than did NRT monotherapy. The recommendation to use combination NRT carries a strength of evidence rating of B. This is because the small number of studies in this analysis contained heterogeneous combination treatments as well as heterogeneous comparison conditions. It is important to note that the FDA has not approved a combination NRT strategy for treatment of smoking cessation. Because there is relatively little safety data on the conjoint use of NRTs, and because combination NRT could increase the risk of nicotine overdose, the panel recommends that this treatment strategy be used only with those patients unable to quit using a single type of pharmacotherapy. Combination NRT also is more expensive than is the use of a single NRT. This extra cost should be considered in making recommendations about NRT use.

Two studies have examined the impact of the combination of the nicotine patch plus nicotine gum on the suppression of the nicotine withdrawal syndrome.[111,116] These studies show that combination NRT is more effective than a single NRT.

It is unknown whether the superiority of combination therapy is due to the use of two types of delivery systems or instead due to the fact that two delivery systems tend to produce higher blood nicotine levels than does the use of a single type of NRT. However, there is only modest evidence that using two forms of passive pharmacotherapies or increasing the dose of a single NRT increases long-term abstinence rates.[117–120] This suggests that the increment in success produced by combination NRTs may depend on the use of two distinct delivery systems: one passive and one *ad libitum*. This conclusion must remain tentative until more research is conducted on this topic. Finally, there is not yet sufficient data to determine whether combination NRTs are particularly efficacious with subpopulations of smokers (eg, those high in nicotine dependence).

Pharmacotherapies Not Recommended by the Guideline Panel

Antidepressants Other Than Bupropion SR and Nortriptyline. Smoking is significantly more prevalent among individuals with a history of depression, and these individuals have more difficulty quitting smoking than do smokers without a history of depression.[121–123] One antidepressant, bupropion SR, has been documented as effective for smoking cessation and approved by the FDA for this use (see above). Nortriptyline also appears to be effective (see above), although the FDA has not approved this medication for treatment of tobacco dependence. Trials have investigated the use of other antidepressants for smoking cessation, including other tricyclics and selective serotonin reuptake inhibitors, but no published articles met selection criteria for review. Because of a paucity of data, the panel drew no conclusions about antidepressant therapy for smoking cessation except to recommend bupropion SR as a first-line agent and nortriptyline as a second-line agent.

Anxiolytics/Benzodiazepines/Beta-Blockers. A few trials have evaluated anxiolytics as a treatment for smoking cessation. Individual trials of propranolol[124] (a beta-blocker) and diazepam[125] (an anxiolytic) did not reveal a beneficial effect for these drugs compared with control interventions. Of the two studies assessing the anxiolytic buspirone that met inclusion criteria, only one revealed evidence of efficacy relative to placebo.[126,127] Because of a lack of data, no meta-analyses were conducted, and no conclusions were drawn regarding the efficacy of anxiolytics in smoking cessation.

Silver Acetate. The two randomized clinical trials[128,129] of silver acetate that met selection criteria revealed no beneficial effects for smoking cessation; therefore, the use of silver acetate as either a primary or an adjunctive treatment for smoking cessation was not supported.

Mecamylamine. Two studies meeting selection criteria evaluated the efficacy of mecamylamine for smoking cessation. In the single study that compared mecamylamine alone to placebo, no efficacy was noted.[130] In both studies, one combination of mecamylamine plus the nicotine patch was compared to placebo; in only one of these studies was the difference significant.[131] Because of these findings, the panel drew no conclusions regarding mecamylamine as a sole medication.

Pharmacotherapy for Treating Tobacco Dependence: Issues Relevant to Use

Overcoming Clinician Reluctance to Use Pharmacotherapy. Some clinicians are reluctant to recommend and prescribe pharmacotherapy for their patients who smoke. Several reasons have been cited for this reluctance, including clinician beliefs that are prevalent but not supported by evidence. Examples of such beliefs are: smoking is a lifestyle choice and not a true dependence disorder; pharmacotherapy should be reserved for heavily dependent smokers or used only in conjunction with an intensive cessation treatment; and smokers will be most successful if they first try to quit on their own. Clinical and epidemiological data strongly counter these beliefs. A variety of findings show that tobacco dependence meets all accepted criteria for a drug dependence disorder. In most users, tobacco use produces tolerance, a well-characterized withdrawal syndrome, and an inability to control future use.[25] Thus, tobacco dependence warrants medical treatment just as do other drug dependence disorders and other chronic diseases.

The panel concluded that, in the vast majority of cases, it is inappropriate to reserve pharmacotherapy until patients have tried to quit on their own. Although many smokers have quit on their own, the vast majority of unaided quit attempts, between 90–95%, end in failure.[2,65] By using the pharmacotherapies found to be effective in this guideline, clinicians can double or triple their patients' chances of abstinence Pharmacotherapies recommended in this guideline have been used effectively with psychosocial treatments that have varied greatly in intensity. When intensity of adjuvant treatments has been examined, data reveal that the pharmacotherapies are effective at low as well as high levels of psychosocial treatment intensity.[132,133] Therefore, clinicians should recommend effective psychosocial treatments such as counseling, in addition to pharmacotherapy, to all patients for whom it is appropriate. Finally, pharmacotherapies are effective for a broad range of smokers, not just "hard core" smokers.

Extended Use of Pharmacotherapy. For some patients, it may be appropriate to continue pharmacotherapeutic treatment (bupropion SR or NRT) for periods longer than usually recommended. The Lung Health Study, which studied almost 4,000 smokers with early evidence COPD, reported that of the sustained quitters, 38% of the women and 30% of the men were still using nicotine gum at 12 months.[134] Other studies also have found that, among patients given free access to nicotine gum, 15–20% of successful abstainers continue to use the gum for a year or longer.[135,136] Although weaning should be encouraged for all smoking cessation pharmacotherapies, continued use of such medication is clearly preferable to a return to smoking with respect to health consequences. This is because, unlike smoking, these medications do not (A) contain non-nicotine toxic substances (eg, "tar" and carbon monoxide), (B) produce dramatic surges in blood nicotine levels, and/or (C) produce strong dependence.[137]

Recommending Specific Pharmacotherapy for Specific Patient Subgroups

There are five FDA-approved medications for treating tobacco dependence. Clinicians are interested in which medications to use with which patients. Unfortunately, this guideline provides little guidance on this topic, because of a lack of relevant research. Some studies have directly compared the efficacies of FDA-approved pharmacotherapies;[118] however, there are too few studies to yield definitive conclusions.

More research is needed before evidence-based pharmacotherapy algorithms can be formulated; however, several factors may guide the clinician in choosing medica-

Table 33. Suggestions for the Clinical Use of Bupropion SR (FDA approved)

Patient selection	Appropriate as a first-line pharmacotherapy for smoking cessation.
Precautions	*Pregnancy:* Pregnant smokers should be encouraged to quit first without pharmacologic treatment. Bupropion SR should be used during pregnancy only if the increased likelihood of smoking abstinence, with its potential benefits, outweighs the risk of bupropion SR treatment and potential concomitant smoking. Similar factors should be considered in lactating women. (FDA Class B) *Cardiovascular diseases:* Generally well tolerated; infrequent reports of hypertension. *Adverse effects:* The most common adverse effects reported by bupropion SR users were insomnia (35–40%) and dry mouth (10%). *Contraindications:* Bupropion SR is contraindicated in individuals with a history of seizure disorder, a history of an eating disorder, who are using another form of bupropion (Wellbutrin or Wellbutrin SR), or who have used a monoamine oxidase inhibitor in the past 14 days.
Dosage	Patients should begin with a dose of 150 mg q AM for 3 days, then increase to 150 mg bid. Dosing at 150 mg bid should continue for 7–12 weeks following the quit date. Unlike nicotine replacement products, patients should begin bupropion SR treatment 1–2 weeks *before* they quit smoking. For maintenance therapy, consider bupropion SR 150 mg bid for up to 6 months.
Availability	Zyban: Prescription only.
Prescribing instructions	*Cessation prior to quit date:* Recognize that some patients will lose their desire to smoke prior to their quit date, or will spontaneously reduce the amount they smoke. *Scheduling of dose:* If insomnia is marked, taking the PM dose earlier (in the afternoon, at least 8 hours after the first dose) may provide some relief. *Alcohol:* Use alcohol only in moderation.
Cost/day*	$3.33

*Cost data is based on the retail price of the medication purchased at a national chain pharmacy located in Madison, Wisconsin, April 2000.
FDA = United States Food and Drug Administration

tions for specific patient subpopulations. For example, highly dependent smokers who use nicotine gum should be urged to use 4 mg, as opposed to 2 mg, gum.[109,110] Also, bupropion SR and nortriptyline have been demonstrated to be efficacious in patients trying to quit who have a history of depression.[138,139] Additionally, for patients concerned about weight gain, some pharmacotherapies (eg, bupropion SR, NRT, in particular nicotine gum) have been shown to delay but not prevent weight gain during their use. Moreover, research suggests that some treatments (eg, NRTs) are less efficacious in women than in men.[140,141] Finally, patient preferences and patient expectations regarding outcome also are important in guiding the choice of a specific pharmacotherapy.[142] A series of clinical recommendations for pharmacotherapy selection is shown in Table 4 in Chapter 3. For a description of some of the issues related to the use of pharmacotherapy with pregnant women, please refer to the Pregnancy section in Chapter 7.

Currently, there is insufficient evidence to suggest that the use of tobacco dependence pharmacotherapies increases long-term abstinence rates among users of smokeless tobacco. Specifically, studies conducted with nicotine gum and the nicotine patch have shown that these two medications have not increased abstinence rates in this population.[143]

Use of Nicotine Replacement Therapy in Cardiovascular Patients. Soon after the nicotine patch was released, the media reported a possible link between the use of this medication and cardiovascular risk. This question has been systematically studied since that time. Separate analyses have now documented the lack of an association between the nicotine patch and acute cardiovascular events[144–146] even in patients who continued to smoke intermittently while on the nicotine patch.[147] Because of inaccurate media coverage in the past, it may be important to inform patients who are reluctant to use NRTs that there is no evidence of increased cardiovascular risk with these medications. Note that package inserts recommend caution with acute cardiovascular diseases (see Tables 33–37).

Future Research. The following pharmacotherapeutic topics require additional research:

- The effectiveness of a nicotine sublingual tablet as a tobacco dependence medication.
- The effectiveness of buspirone as a tobacco dependence medication.
- The effectiveness of mecamylamine as a tobacco dependence medication, both used alone and in combination with other medications.
- The use of sustained or long-term pharmacotherapy for treating tobacco dependence.
- The use of other antidepressants or anxiolytics as tobacco dependence medications.
- The relative efficacy and safety of the five FDA-approved pharmacotherapies both, in general, and for spe-

Table 34. Suggestions for the Clinical Use of Nicotine Gum (FDA approved)

Patient selection	Appropriate as a first-line pharmacotherapy for smoking cessation.
Precautions	*Pregnancy:* Pregnant smokers should be encouraged to quit first without pharmacologic treatment. Nicotine gum should be used during pregnancy only if the increased likelihood of smoking abstinence, with its potential benefits, outweighs the risk of nicotine replacement and potential concomitant smoking. Similar factors should be considered in lactating women. (FDA Class D) *Cardiovascular diseases:* NRT is not an independent risk factor for acute myocardial events. NRT should be used with caution among particular cardiovascular patient groups: those in the immediate (within 2 weeks) postmyocardial infarction period, those with serious arrhythmias, and those with serious or worsening angina pectoris. *Adverse effects:* Common adverse effects of nicotine chewing gum include mouth soreness, hiccups, dyspepsia, and jaw ache. These effects are generally mild and transient, and often can be alleviated by correcting the patient's chewing technique (see prescribing instructions below).
Dosage	Nicotine gum is available in 2 mg and 4 mg (per piece) doses. The 2 mg gum is recommended for patients smoking less than 25 cigarettes per day, while the 4 mg gum is recommended for patients smoking 25 or more cigarettes per day. Generally, the gum should be used for up to 12 weeks with no more than 24 pieces/d. Clinicians should tailor the dosage and duration of therapy to fit the needs of each patient.
Availability	Nicorette, Nicorette Mint: over-the-counter only.
Prescribing instructions	*Chewing technique:* Gum should be chewed slowly until a "peppery" or "minty" taste emerges, then "parked" between cheek and gum to facilitate nicotine absorption through the oral mucosa. Gum should be slowly and intermittently "chewed and parked" for about 30 minutes or until the taste dissipates. *Absorption:* Acidic beverages (eg, coffee, juices, soft drinks) interfere with the buccal absorption of nicotine, so eating and drinking anything except water should be avoided for 15 minutes before and during chewing. *Scheduling of dose:* Patients often do not use enough gum to get the maximum benefit: they chew too few pieces per day and they do not use the gum for a sufficient number of weeks. Instructions to chew the gum on a fixed schedule (at least one piece every 1–2 hours) for at least 1–3 months may be more beneficial than ad libitum use.
Cost/day*	$6.25 for ten 2 mg pieces. $6.87 for ten 4 mg pieces.

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*Cost data is based on the retail price of the medication purchased at a national chain pharmacy located in Madison, Wisconsin, April 2000.

FDA = United States Food and Drug Administration. NRT = nicotine replacement therapy

Table 35. Suggestions for Clinical Use of the Nicotine Inhaler (FDA approved)

Patient selection	Appropriate as a first-line pharmacotherapy for smoking cessation.
Precautions	*Pregnancy:* Pregnant smokers should be encouraged to quit first without pharmacologic treatment. The nicotine inhaler should be used during pregnancy only if the increased likelihood of smoking abstinence, with its potential benefits, outweighs the risk of nicotine replacement and potential concomitant smoking. Similar factors should be considered in lactating women. (FDA Class D) *Cardiovascular diseases:* NRT is not an independent risk factor for acute myocardial events. NRT should be used with caution among particular cardiovascular patient groups: those in the immediate (within 2 weeks) postmyocardial infarction period, those with serious arrhythmias, and those with serious or worsening angina pectoris. *Local irritation reactions:* Local irritation in the mouth and throat was observed in 40% of patients using the nicotine inhaler. Coughing (32%) and rhinitis (23%) also were common. Severity was generally rated as mild and the frequency of such symptoms declined with continued use.
Dosage	A dose from the nicotine inhaler consists of a puff or inhalation. Each cartridge delivers 4 mg of nicotine over 80 inhalations. Recommended dosage is 6–16 cartridges/day. Recommended duration of therapy is up to 6 months. Instruct patient to taper dosage during the final 3 months of treatment.
Availability	Nicotrol Inhaler: prescription only.
Prescribing instructions	*Ambient temperature:* Delivery of nicotine from the inhaler declines significantly at temperatures below 40°F. In cold weather the inhaler and cartridges should be kept in an inside pocket or warm area. *Duration:* Use is recommended for up to 6 months, with gradual reduction in frequency of use over the last 6–12 weeks of treatment. *Absorption:* Acidic beverages (eg, coffee, juices, soft drinks) interfere with the buccal absorption of nicotine, so eating and drinking anything except water should be avoided for 15 minutes before and during inhalation. *Best effects:* Best effects are achieved by frequent puffing.
Cost/day*	$10.94 for 10 cartridges.

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*Cost data is based on the retail price of the medication purchased at a national chain pharmacy located in Madison, Wisconsin, April 2000.

FDA = United States Food and Drug Administration. NRT = nicotine replacement therapy

Table 36. Suggestions for Clinical Use of Nicotine Nasal Spray (FDA approved)

Patient selection	Appropriate as a first-line pharmacotherapy for smoking cessation.
Precautions	*Pregnancy:* Pregnant smokers should be encouraged to quit first without pharmacologic treatment. Nicotine nasal spray should be used during pregnancy only if the increased likelihood of smoking abstinence, with its potential benefits, outweighs the risk of nicotine replacement and potential concomitant smoking. Similar factors should be considered in lactating women. (FDA Class D) *Cardiovascular diseases:* NRT is not an independent risk factor for acute myocardial events. NRT should be used with caution among particular cardiovascular patient groups: those in the immediate (within 2 weeks) postmyocardial infarction period, those with serious arrhythmias, and those with serious or worsening angina pectoris. *Nasal/airway reactions:* Some 94% of users report moderate to severe nasal irritation in the first 2 days of use; 81% still reported nasal irritation after 3 weeks, although rated severity was mild to moderate. Nasal congestion and transient changes in sense of smell and taste also were reported. Nicotine nasal spray should not be used in persons with severe reactive airway disease. *Dependency:* Nicotine nasal spray has a dependence potential intermediate between other nicotine-based therapies and cigarettes. About 15–20% of patients report using the active spray for longer periods than recommended (6–12 months), and 5% used the spray at a higher dose than recommended.
Dosage	A dose of nicotine nasal spray consists of one 0.5 mg delivery to each nostril (1 mg total). Initial dosing should be 1–2 doses per hour, increasing as needed for symptom relief. Minimum recommended treatment is 8 doses/d, with a maximum limit of 40 doses/d (5 doses/h). Each bottle contains approximately 100 doses. Recommended duration of therapy is 3–6 months.
Availability	Nicotrol NS: Prescription only.
Prescribing instructions	*Dose delivery:* Patients should not sniff, swallow, or inhale through the nose while administering doses because this increases irritating effects. The spray is best delivered with the head tilted slightly back.
Cost/day*	$5.40 for 12 doses.

*Cost data is based on the retail price of the medication purchased at a national chain pharmacy located in Madison, Wisconsin, April 2000.
FDA = United States Food and Drug Administration. NRT = nicotine replacement therapy.

cific subpopulations (eg, women, adolescents, smokeless users, depressed patient, post-myocardial infarction patients).

- The use of combined tobacco dependence pharmacotherapies in general and for specific subpopulations (eg, highly dependent smokers).
- The efficacy of combining active and passive NRTs versus combining two active or two passive NRTs.
- When clonidine and nortriptyline should be used in lieu of or in combination with other tobacco dependence pharmacotherapies.
- The optimal combination of counseling and pharmacotherapy intensities (see Tables 38 and 39).

Over-the-Counter Pharmacotherapeutic Interventions. Recommendation: Over-the-counter nicotine patch therapy is more efficacious than placebo and its use should be encouraged. (Strength of evidence = B)

There were three placebo-controlled studies with six arms that met selection criteria for the analysis of pharmacotherapeutic interventions in over-the-counter (OTC) settings. These three studies specifically examined the effect of patch versus placebo The only adjuvant treatments in these studies were a self-help manual, instructions contained in the package, or written directions for using the patch. As shown in Table 40, the use of the nicotine patch in OTC settings nearly doubles abstinence rates, compared with placebo. There were too few studies done in the OTC setting to permit meta-analysis of the OTC effect of any other pharmacotherapy.

The FDA has approved nicotine gum and the nicotine patch for OTC use. These products are identical to the patches and gum previously available only via prescription. Although the OTC status of these medications has increased their availability and use, this does not reduce the clinician's responsibility to intervene with smokers or insurers/managed care organizations to cover the costs of such treatment. Moreover, OTC availability may enhance the capacity of nonphysician clinicians to intervene comprehensively when treating tobacco dependence.

All clinicians have specific responsibilities regarding these products, such as encouraging their use when appropriate, providing counseling and follow-up, encouraging total abstinence, and offering instruction on appropriate use. Additionally, patients should be urged to read the package insert and consult with their pharmacist. Finally, the clinician may advise patients regarding the selection and use of an OTC product versus a non-OTC product such as bupropion SR or a prescription nicotine replacement treatment (nasal spray or inhaler). Clinicians also may provide or recommend counseling for patients quitting with an OTC product. It should be noted that a single recent study not included within the meta-analysis reported low abstinence rates with OTC patch use.[148]

Table 37. Suggestions for Clinical Use of the Nicotine Patch (FDA approved)

Patient selection	Appropriate as a first-line pharmacotherapy for smoking cessation.	
Precautions	*Pregnancy:* Pregnant smokers should be encouraged to quit first without pharmacologic treatment. The nicotine patch should be used during pregnancy only if the increased likelihood of smoking abstinence, with its potential benefits, outweighs the risk of nicotine replacement and potential concomitant smoking. Similar factors should be considered in lactating women. (FDA Class C)	
	Cardiovascular diseases: NRT is not an independent risk factor for acute myocardial events. NRT should be used with caution among particular cardiovascular patient groups: those in the immediate (within 2 weeks) postmyocardial infarction period, those with serious arrhythmias, and those with serious or worsening angina pectoris.	
	Skin reactions: Up to 50% of patients using the nicotine patch will have a local skin reaction. Skin reactions are usually mild and self-limiting, but may worsen over the course of therapy. Local treatment with hydrocortisone cream (1%) or triamcinolone cream (0.5%) and rotating patch sites may ameliorate such local reactions. In less than 5% of patients such reactions require the discontinuation of nicotine patch treatment.	
	Other adverse effects: insomnia.	
Dosage	Treatment of 8 weeks or less has been shown to be as efficacious as longer treatment periods. 16-hour and 24-hour patches are of comparable efficacy. Clinicians should consider individualizing treatment based on specific patient characteristics such as previous experience with the patch, amount smoked, degree of addictiveness, etc. Finally, clinicians should consider starting treatment on a lower patch dose in patients smoking 10 or fewer cigarettes per day.	
Availability	Nicoderm CQ, Nicotrol, generic: over-the-counter.	
	Nicotine patches, generic (various doses): prescription.	
Brand	Duration	Dosage
Nicoderm CQ	4 weeks	21 mg/24 hours
	then 2 weeks	14 mg/24 hours
	then 2 weeks	7 mg/24 hours
Nicotrol	8 weeks	15 mg/16 hours
Prescribing instructions	*Location:* At the start of each day the patient should place a new patch on a relatively hairless location, typically between the neck and waist.	
	Activities: No restrictions while using the patch.	
	Time: Patches should be applied as soon as the patient wakes on their quit day. With patients who experience sleep disruption, have the patient remove the 24-hour patch prior to bedtime or use the 16-hour patch.	
Cost/day*	Brand name patches (Nicoderm CQ, Nicotrol): $4.00–$4.50.	
	Generic patches recently became available and may be less expensive.	

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*Cost data is based on the retail price of the medication purchased at a national chain pharmacy located in Madison, Wisconsin, April 2000.

FDA = United States Food and Drug Administration. NRT = nicotine replacement therapy.

Future Research Important topics for future research are:

- The efficacy of nicotine patch and nicotine gum when access is OTC.
- The extent to which individuals use pharmacotherapies optimally when access is OTC.
- The extent to which the efficacy of OTC pharmacotherapy is enhanced by adjuvant treatments (eg, pharmacist counseling, telephone counseling, computer self-help resources, clinician interventions).
- The extent to which OTC status increases or reduces the use of pharmacotherapies by poor or minority populations.

7. Special Populations

Background

Many factors could potentially affect the choice, delivery, and efficacy of tobacco dependence treatments. For instance, should interventions be tailored or modified on the basis of gender, race/ethnicity, age, comorbidity, or hospitalization status? Should pregnant smokers receive pharmacotherapy? Do tobacco dependence interventions interfere with other chemical dependence treatments? These and other special issues and populations are considered in this chapter.

A variety of health care specialties can play a key role in addressing issues related to special populations (eg, obstetricians and family practitioners for pregnant smokers; gynecologists and family practitioners for preconception counseling and general health maintenance; pediatricians, family practitioners, and dentists for children and adolescents; internists, including cardiologists, pulmonologists, oncologists, and general internists, and family practitioners for hospitalized patients; geriatricians for older smokers; and dentists and dental hygienists for smokeless tobacco users).

Table 38. Suggestions for Clinical Use of Clonidine (not FDA approved for smoking cessation)

Patient selection	Appropriate as a second-line pharmacotherapy for smoking cessation.
Precautions	*Pregnancy:* Pregnant smokers should be encouraged to quit first without pharmacologic treatment. Clonidine should be used during pregnancy only if the increased likelihood of smoking abstinence, with its potential benefits, outweighs the risk of clonidine and potential concomitant smoking and first-line pharmacotherapies have not been successful. Similar factors should be considered in lactating women. (FDA Class C) *Adverse effects:* Most commonly reported adverse effects include dry mouth (40%), drowsiness (33%), dizziness (16%), sedation (10%), and constipation (10%). As an antihypertensive medication, clonidine can be expected to lower blood pressure in most patients. Therefore, clinicians may need to monitor blood pressure when using this medication. *Rebound hypertension:* Failure to gradually reduce the dose over a period of 2–4 days may result in a rapid increase in blood pressure, agitation, confusion, and/or tremor.
Dosage	Doses used in various clinical cessation trials have varied significantly, from 0.15–0.75 mg/d PO to 0.10–0.20 mg/d transdermal (TTS), without a clear dose-response relation to cessation. Initial dosing is typically 0.10 mg bid PO or 0.10 mg/d TTS, increasing by 0.10 mg/d per week if needed. The dose duration also varied across the clinical trials, ranging from 3 weeks to 10 weeks.
Availability	*Oral:* Clonidine (generic), Catapres: prescription only. *Transdermal:* Catapres: prescription only.
Prescribing instructions	*Initiate:* Initiate clonidine shortly before (up to 3 days) or on the quit date. *Location (TTS Only):* At the start of each week, the patient should place a new patch on a relatively hairless location between the neck and waist. *Activities:* Use of either form may produce sedation, a hazard while driving or operating machinery. Users should not discontinue clonidine therapy abruptly.
Cost/day*	Clonidine: $0.24 for 0.2 mg. Catapres (transdermal): $3.50.

*Cost data is based on the retail price of the medication purchased at a national chain pharmacy located in Madison, Wisconsin, April 2000.
FDA = United States Food and Drug Administration.

Table 39. Suggestions for Clinical Use of Nortriptyline (FDA approved)

Patient selection	Appropriate as a second-line pharmacotherapy for smoking cessation.
Precautions	*Pregnancy:* Pregnant smokers should be encouraged to quit first without pharmacologic treatment. Nortriptyline should be used during pregnancy only if the increased likelihood of smoking abstinence, with its potential benefits, outweighs the risk of nortriptyline, potential concomitant smoking, and first-line pharmacotherapies have not been successful. Nortriptyline has been associated with limb reduction anomalies. *Adverse effects:* Most commonly reported adverse effects include sedation, dry mouth (64–78%), blurred vision (16%), urinary retention, lightheadedness (49%), and shaky hands (23%). *Cardiovascular effects:* Because of risk of arrhythmias, changes in contractility, and blood flow, use with extreme caution in patients with cardiovascular disease.
Dosage	Doses used in smoking cessation trials have initiated treatment at a dose of 25 mg/d, increasing gradually to a target dose of 75–100 mg/d. Duration of treatment used in smoking cessation trials has been approximately 12 weeks.
Availability	Nortriptyline HCl (generic): prescription only.
Prescribing instructions	Therapy is initiated 10–28 days before the quit date to allow nortriptyline to reach steady state at the target dose. *Activities:* Use may produce sedation, a hazard while driving or operating machinery. Overdose may produce marked cardiotoxic effects. Risk of overdose should be considered carefully in using nortriptyline.
Cost/day*	$0.74 for 75 mg

*Cost data is based on the retail price of the medication purchased at a national chain pharmacy located in Madison, Wisconsin, April 2000.
FDA = United States Food and Drug Administration. NRT = nicotine replacement therapy.

One overriding issue relevant to all tobacco users considering a quit attempt is to ensure that all textual materials used (eg, self-help brochures) are at an appropriate reading level. This is particularly important given epidemiological data showing that tobacco use rates are markedly higher among individuals of lower educational attainment.[149]

Gender

Recommendation: The same smoking cessation treatments are effective for both men and women. Therefore, except in the case of the pregnant smoker, the

Table 40. Meta-Analysis: Efficacy of and Estimated Abstinence Rates for Over-the-Counter Nicotine Patch Therapy (*n* = 3 studies)

OTC Therapy	Number of Arms	Estimated Odds Ratio (95% CI)	Estimated Abstinence Rate (95% CI)
Placebo	3	1.0	6.7
Over-the-counter nicotine patch therapy	3	1.8 (1.2, 2.8)	11.8 (7.5, 16.0)

OTC = over-the-counter. CI = confidence interval.

Table 41. Meta-Analysis: Efficacy of and Estimated Abstinence Rates for Augmented Interventions with Pregnant Smokers (*n* = 7 studies)

Pregnant Smokers	Number of Arms	Estimated Odds Ratio (95% CI)	Estimated Abstinence Rate (95% CI)
Usual care	7	1.0	6.6
Augmented intervention	8	2.8 (2.2, 3.7)	16.8 (13.1, 20.5)

CI = confidence interval

same interventions can be used with both men and women. (Strength of Evidence = B)

One important question regarding quitting smoking is whether men and women should receive different cessation interventions. Smoking cessation clinical trials reveal that the same treatments benefit both men and women;[140,150] however, research suggests that some treatments are less efficacious in women than in men (eg, NRTs).[151,152]

Although research suggests that women benefit from the same interventions as do men, women may face different stressors and barriers to quitting that may be addressed in treatment. These include greater likelihood of depression, greater weight control concerns, hormonal cycles, and others.[153] This suggests that women may benefit from tobacco dependence treatments that address these topics, although few studies have examined programs targeted to one gender. Finally, women who are considering becoming pregnant may be especially receptive to tobacco dependence treatment.

Future Research. The following topics regarding gender require additional research:

- The efficacy of interventions that are targeted to specific genders.
- The impact of gender-specific motives that may increase quit attempts and success (eg, quitting to improve fertility and reproductive health, erectile dysfunction, pregnancy outcomes, physical appearance, and osteoporosis).
- Gender differences in efficacy of tobacco dependence treatments found to be effective in this guideline.

Pregnancy

Recommendation: Because of the serious risks of smoking to the pregnant smoker and the fetus, whenever possible pregnant smokers should be offered extended or augmented psychosocial interventions that exceed minimal advice to quit. (Strength of Evidence = A)

Recommendation: Although abstinence early in pregnancy will produce the greatest benefits to the fetus and expectant mother, quitting at any point in pregnancy can yield benefits. Therefore, clinicians should offer effective smoking cessation interventions to pregnant smokers at the first prenatal visit as well as throughout the course of pregnancy. (Strength of Evidence = B)

Recommendation: Pharmacotherapy should be considered when a pregnant woman is otherwise unable to quit, and when the likelihood of quitting, with its potential benefits, outweighs the risks of the pharmacotherapy and potential continued smoking. (Strength of Evidence = C)

The selection criteria for the pregnancy meta-analysis were adjusted to reflect this unique population. Abstinence data were included only if they were biochemically confirmed, because of reports of high levels of deception regarding smoking status among pregnant women.[154–156] Studies that had follow-up time points of less than 5 months were included because of the desire for preparturition data. For the meta-analysis, either minimal interventions (< 3 min) or interventions labeled as "usual care" constituted the reference condition. Seven studies met the criteria and were included in the analysis comparing augmented smoking cessation interventions with usual care in pregnant women. A "usual care" intervention with pregnant smokers typically consists of a recommendation to stop smoking, often supplemented by provision of self-help material or referral to a stop-smoking program. Extended or augmented psychosocial interventions typically involve these treatment components as well as more intensive counseling than minimal advice. As can be seen from the data in Table 41, extended or augmented interventions are significantly more efficacious than usual care in pregnant women.

Components of some extended or augmented psychosocial interventions are listed in Table 42. These interventions were selected from articles included in the Table 41 meta-analysis and should guide the clinician treating the pregnant smoker.

Smoking in pregnancy imparts risks to both the woman and the fetus. Cigarette smoking by pregnant women has been shown to cause adverse fetal outcomes, including stillbirth, spontaneous abortion, decreased fetal growth, premature birth, low birthweight, placental abruption, sud-

Table 42. Examples of Effective Interventions with Pregnant Patients

Ershoff et al (1989)[154]	Brief health educator discussion of risks (3–5 min); advised of a free smoking cessation class; and pregnancy-specific self-help materials mailed weekly for 7 weeks.
Walsh et al (1997)[155]	Physician advice regarding risks (2–3 min); videotape with information on risks, barriers, and tips for quitting; midwife counseling in one 10-minute session; self-help manual; and follow-up letters.
Windsor et al (1985)[156]	Pregnancy-specific self-help materials (Pregnant Woman's Self-Help Guide To Quit Smoking) and one 10-minute counseling session with a health educator.
Windsor et al (1993)[37]	Cessation skills and risk counseling in one 15-minute session by a health counselor; education on how to use pregnancy-specific self-help materials (same materials as in Windsor et al, 1985[156]); a follow-up medical letter; and social support with a buddy letter, a buddy contract, and a buddy tip sheet.

den infant death syndrome, cleft palate and cleft lip, and childhood cancer. Many women are motivated to quit during pregnancy, and health care professionals can take advantage of this motivation by reinforcing the knowledge that cessation will reduce health risks to the fetus and that there are postpartum benefits for both the mother and child.[157]

The first step in intervention is assessment of tobacco use status. This is especially important in a population with reported high rates of deception. Research has shown that the use of multiple choice questions (see Table 43), as opposed to a simple yes/no question can increase disclosure among pregnant women by as much as 40%.[158]

Quitting smoking prior to conception or early in the pregnancy is most beneficial, but health benefits result from abstinence at any time. Therefore, a pregnant smoker should receive encouragement and assistance in quitting throughout her pregnancy.

Even women who have maintained total abstinence from tobacco for 6 or more months during pregnancy have a high rate of relapse in the postpartum period.[159,160] Postpartum relapse may be decreased by continued emphasis on the relationship between maternal smoking and poor health outcomes in infants and children (sudden infant death syndrome, respiratory infections, asthma, and middle ear disease). [159–162] Preventing postpartum relapse is, however, an area that would benefit from future research. Table 43 outlines clinical factors to address when counseling pregnant women about smoking.

For pregnant smokers who are unable to quit with the help of an augmented intervention (see Table 42), clinicians may consider additional or alternative psychosocial treatments such as those described in Chapter 4. The exception to this would be the use of rapid smoking, which can result in extremely high blood nicotine levels.

Clinicians may choose to consider pharmacotherapy for pregnant smokers who have been unable to quit using psychosocial interventions. In such cases, the clinician and pregnant smoker must contrast the risks and unknown efficacy of pharmacotherapy in pregnant women with the substantial risks of continued smoking. Although smoking during pregnancy clearly leads to substantial risks for both the pregnant smoker and the fetus, the clinician and patient also must be aware of potential risks of different pharmacotherapies. For example, a number of studies have shown that nicotine itself presents risks to the fetus, including neurotoxicity,[163] and bupropion SR has been shown to cause seizures in 1 out of 1,000 patients.[164]

If the clinician and pregnant or lactating patient decide to use NRT pharmacotherapy, the clinician should consider monitoring blood nicotine levels to assess level of drug delivery. In addition, the clinician should consider using medication doses that are at the low end of the effective dose range, and consider choosing delivery systems that yield intermittent, rather than continuous, drug exposure (eg, nicotine gum rather than the nicotine patch). Because none of these medications has been tested in pregnant women for efficacy in treating tobacco dependence, the relative ratio of risks to benefits is unclear. Additionally, since small amounts of these medications are passed through breast milk, they may pose some risks for nursing infants.

Future Research. The following topics regarding smoking and pregnancy require additional research:

- Relapse prevention with pregnant women and women who have recently given birth.
- The efficacy of relapse prevention programs for spontaneous "self-quitters."
- The most efficacious amount of contact time, number of sessions, and duration for smoking cessation interventions with pregnant women.
- The efficacy of various counseling and behavioral therapies and motivational interventions (eg, physiologic feedback of adverse impacts, quitting benefits).
- Efficacious treatments for highly dependent smokers.
- The safety and efficacy of tobacco dependence pharmacotherapy during pregnancy to the woman and the fetus, including the relative risks and benefits of pharmacotherapy use as a function of dependence and the appropriate formulation and timing of pharmacotherapy
- The safety and efficacy of tobacco dependence pharmacotherapy to the woman and child during nursing.
- The efficacy of targeted or individualized interventions in pregnancy.

Table 43. Clinical Practice When Assisting a Pregnant Patient in Smoking Cessation

Clinical Practice	Rationale
Assess pregnant woman's tobacco use status using a multiple-choice question to improve disclosure.	Many pregnant women deny smoking, and the multiple-choice question format improves disclosure. For example: Which of the following statements best describes your cigarette smoking? • I smoke regularly now—about the same as before finding out I was pregnant. • I smoke regularly now, but I've cut down since I found out I was pregnant. • I smoke every once in a while. • I have quit smoking since finding out I was pregnant. • I wasn't smoking around the time I found out I was pregnant, and I don't currently smoke cigarettes.
Congratulate those smokers who have quit on their own.	To encourage continued abstinence.
Motivate quit attempts by providing educational messages about the impact of smoking on both the woman's and the fetus' health.	These are associated with higher quit rates.
Give clear, strong advice to quit as soon as possible.	Quitting early in pregnancy provides the greatest benefit to the fetus.
Suggest the use of problem-solving methods and provide social support and pregnancy-specific self-help materials.	Reinforces pregnancy-specific benefits and ways to achieve cessation.
Arrange for follow-up assessments throughout pregnancy, including further encouragement of cessation.	The woman and her fetus will benefit even when quitting occurs late in pregnancy.
In the early postpartum period, assess for relapse and use relapse prevention strategies recognizing that patients may minimize or deny tobacco use.	Postpartum relapse rates are high even if a woman maintains abstinence throughout pregnancy. Relapse prevention may start during pregnancy (see Chapter 3C, Brief Strategies C1 and C2).

- Strategies for linking preconception, pregnancy, and postpartum (including pediatric) interventions.

Racial and Ethnic Minorities

Recommendation: Smoking cessation treatments have been shown to be effective across different racial and ethnic minorities. Therefore, members of racial and ethnic minorities should be provided treatments shown to be effective in this guideline. (Strength of Evidence = A)

Recommendation: Whenever possible, tobacco dependence treatments should be modified or tailored to be appropriate for the ethnic or racial populations with which they are used. (Strength of Evidence = C)

Ethnic and racial minority groups in the United States—African Americans, American Indians/Alaska Natives, Asians and Pacific Islanders, Hispanics—experience higher mortality in a number of disease categories, compared with others. For example, African Americans experience substantial excess mortality from cancer, cardiovascular disease, and infant death, all of which are directly affected by tobacco use.[165,166] American Indian/Alaska Native subgroups have some of the highest documented rates of infant mortality caused by sudden infant death syndrome,[167] which also is affected by tobacco use. Therefore, there is a critical need to deliver effective tobacco dependence interventions to ethnic and racial minorities Unfortunately, there is evidence that large proportions of some racial/ethnic groups lack adequate access to primary care providers.[166] This suggests that special efforts and resources should be provided to meet the treatment needs of these populations.

There are well-documented differences between racial and ethnic minorities and whites in smoking prevalence, smoking patterns, and quitting behavior in the United States.[166,168,169] In addition, smoking prevalence and patterns vary substantially among minority subgroups.[166,167] Racial and ethnic minority groups also differ from whites in awareness of the health effects of smoking[170] and report a sense of fatalism that may affect disease prevention efforts. On the other hand, both tobacco dependence and desire to quit appear to be prevalent across all racial and ethnic groups.[166,168,169,171]

Studies have demonstrated the efficacy of a variety of smoking cessation interventions in minority populations. Nicotine patch,[172] clinician advice,[173,174] counseling,[175] tailored self-help manuals and materials, and telephone counseling[173,176] have been shown to be effective with African Americans. Nicotine patch[177] and self-help materials, including a mood management component,[178] have been shown to be effective with Hispanic smokers. Screening for tobacco use, clinician advice, clinic staff reinforcement, and follow-up materials have been shown to be effective for American Indian populations.[179]

Few studies have examined interventions specifically designed for particular ethnic or racial groups, and there is no consistent evidence that targeted cessation programs result in higher quit rates in these groups than do generic interventions of comparable intensity.[176] Moreover, smoking cessation interventions developed for the general population have been effective with racial and ethnic minority participants. Therefore, clinicians should offer treatments identified as effective in this guideline to their patients from all racial and ethnic groups. It is essential, however, that cessation counseling or self-help materials be conveyed in a language understood by the smoker. Additionally, culturally appropriate models or examples may increase the smoker's acceptance of treatment. Clinicians should remain sensitive to individual differences and health beliefs that may affect treatment acceptance and success in all populations (see section in Chapter 6A, Specialized Assessment).

Future Research. The following topics regarding racial and ethnic minorities require additional research:

- The efficacy of targeted versus generic interventions for different racial and ethnic minority populations.
- The identification of the specific barriers or impediments to treatment or treatment success (eg, socioeconomic status, inadequate access to medical care), and the differential health effects related to smoking patterns for racial and ethnic minorities.
- Motivators of cessation that are especially effective with members of racial and ethnic minorities (eg, fear of illness requiring long-term care and disability).

Hospitalized Smokers

Recommendation: Smoking cessation treatments have been shown to be effective for hospitalized patients. Therefore, hospitalized patients should be provided smoking cessation treatments shown to be effective in this guideline. (Strength of Evidence = B)

Four studies met the selection criteria and were relevant to the analysis comparing augmented smoking cessation treatment with usual care for hospitalized patients. Because the analysis was limited to four studies, no attempt was made to categorize the augmented treatment with respect to intensity or type for the purpose of the meta-analysis. For reference only, the augmented interventions in the analyzed studies included elements such as self-help via brochure or audio/videotape, chart prompt reminding physician to advise smoking cessation, pharmacotherapy, hospital counseling, and postdischarge counseling telephone calls. As can be seen from the data in Table 44, augmented smoking cessation interventions among hospitalized patients increase rates of smoking abstinence

Table 44. Meta-Analysis: Efficacy of and Estimated Abstinence Rates for Augmented Interventions with Hospitalized Smokers ($n = 4$ studies)

Hospitalized Smokers	Number of Arms	Estimated Odds Ratio (95% CI)	Estimated Abstinence Rate (95% CI)
Usual care	4	1.0	19.2
Augmented intervention	6	1.3 (1.04, 1.6)	23.3 (19.5, 27.1)

CI = confidence interval

Table 45. Suggested Interventions for Hospitalized Patients

For every hospitalized patient, the following steps should be taken:

- Ask each patient on admission if he or she uses tobacco and document tobacco use status.
- For current tobacco users, list tobacco use status on the admission problem list and as a discharge diagnosis.
- Use counseling and pharmacotherapy to assist all tobacco users to maintain abstinence and to treat withdrawal symptoms.
- Provide advice and assistance on how to quit during hospitalization and remain abstinent after discharge.

It is vital that hospitalized patients attempt to quit smoking, because smoking may interfere with their recovery. Among cardiac patients, second heart attacks are more common in those who continue to smoke.[16,180] Lung, head, and neck cancer patients who are successfully treated, but who continue to smoke, are at elevated risk for a second cancer.[181–185] Additionally, smoking negatively affects bone and wound healing.[186–188]

Hospitalized patients may be particularly motivated to make a quit attempt, for two reasons. First, the illness resulting in hospitalization may have been caused or exacerbated by smoking, highlighting the patient's personal vulnerability to the health risks of smoking. Second, every hospital in the United States must now be smoke free if it is to be accredited by the Joint Commission on Accreditation of Health Care Organizations. As a result, every hospitalized smoker is temporarily housed in a smoke-free environment. For these reasons, clinicians should use hospitalization as an opportunity to promote smoking cessation in their patients who smoke.[189,190] Patients in long-term care facilities also should receive tobacco dependence interventions identified as efficacious in this guideline. Suggested interventions for hospitalized patients can be found in Table 45.

Future Research. The following topics regarding hospitalized patients require additional research:

- The efficacy of interventions provided by different hospital personnel, including nurses.
- The efficacy of pharmacotherapy with hospitalized patients.
- Relapse prevention once the patient leaves the hospital.

Smokers With Psychiatric Comorbidity and/or Chemical Dependence

Recommendation: Smokers with comorbid psychiatric conditions should be provided smoking cessation treatments identified as effective in this guideline. (Strength of Evidence = C)

Recommendation: Bupropion SR and nortriptyline, efficacious treatments for smoking cessation in the general population, also are effective in treating depression. Therefore, bupropion SR and nortriptyline should be especially considered for the treatment of tobacco dependence in smokers with current or past history of depression. (Strength of Evidence = C)

Recommendation: Evidence indicates that smoking cessation interventions do not interfere with recovery from chemical dependence. Therefore, smokers receiving treatment for chemical dependence should be provided smoking cessation treatments shown to be effective in this guideline, including both counseling and pharmacotherapy (Strength of Evidence = C)

The term "psychiatric comorbidity" refers to the cooccurrence of smoking with another psychiatric disorder. Although it is not necessary to assess for psychiatric comorbidity prior to initiating tobacco dependence treatment, psychiatric comorbidity is important to the assessment and treatment of smokers for several reasons:

- Psychiatric disorders are more common among smokers than in the general population. For instance, as many as 30% of patients seeking smoking cessation services may have a history of depression,[121] and 20% or more may have a history of alcohol abuse or dependence.[191–195] Among abusers of alcohol and drugs, smoking occurs at rates well above population average (eg, greater than 70%).[196–198] These individuals may infrequently present themselves for tobacco dependence treatment. However, such treatments could be conveniently delivered within the context of chemical dependence clinics.
- Smoking cessation or nicotine withdrawal may exacerbate a patient's comorbid condition. For instance, smoking cessation may elicit or exacerbate depression among patients with a prior history of affective disorder.[199–202]
- As noted in the Specialized Assessment section in Chapter 6A, smokers with psychiatric comorbidities have heightened risk for relapse to smoking after a cessation attempt.[88,191,195,202]

Although psychiatric comorbidity places smokers at increased risk for relapse, such smokers can be helped by smoking cessation treatments.[195,203–207] Currently, there is insufficient evidence to determine whether smokers with psychiatric comorbidity benefit more from specialized or tailored cessation treatments than from standard treatments.[102,208] Even though some smokers may experience exacerbation of a comorbid condition upon quitting smoking, most evidence suggests that abstinence entails little adverse impact. For instance, patients in inpatient psychiatric units are able to stop smoking with few adverse effects (eg, little increase in aggression).[209–211] Finally, stopping smoking may affect the pharmacokinetics of certain psychiatric medications.[212] Therefore, clinicians may wish to monitor closely the actions or side effects of psychiatric medications in smokers making a quit attempt.

Evidence shows that bupropion SR is efficacious for both depression and smoking cessation. Therefore, it appears to be an appropriate medication to use with depressed smokers trying to quit. Nortriptyline is also efficacious for both depression and smoking cessation, but its adverse effect profile renders it a second-line medication.

The treatment of tobacco dependence can be provided concurrent to treating patients for other chemical dependencies (alcohol and other drugs). With patients in treatment for chemical dependence, there is little evidence that patients with other chemical dependencies relapse to other drug use when they stop smoking.[209,213,214] However, such patients should be followed closely after they stop smoking.

Future Research. The following topics regarding psychiatric comorbidity and/or chemical dependence require additional research:

- The relative efficacy of bupropion SR and nortriptyline versus NRT in patients with psychiatric comorbidity, including depression.
- The efficacy and impact of tobacco dependence treatments within the context of other chemical dependence treatments.
- The importance and efficacy of specialized assessment and tailored interventions in these populations.

Children and Adolescents

Recommendations: Clinicians should screen pediatric and adolescent patients, and their parents, for tobacco use and provide a strong message regarding the importance of totally abstaining from tobacco use. (Strength of Evidence = C)

Recommendation: Counseling and behavioral interventions shown to be effective with adults should be considered for use with children and adolescents. The content of these interventions should be modified to be developmentally appropriate. (Strength of Evidence = C)

Recommendation: When treating adolescents, clinicians may consider prescriptions for bupropion SR or NRT when there is evidence of nicotine dependence and desire to quit tobacco use. (Strength of Evidence = C)

Recommendation: Clinicians in a pediatric setting should offer smoking cessation advice and interventions to parents to limit children's exposure to second-hand smoke. (Strength of Evidence = B)

Tobacco use is a pediatric concern. In the United States, more than 6,000 children and adolescents try their first cigarette each day.[7] More than 3,000 children and adolescents become daily smokers every day,[8] resulting in approximately 1.23 million new smokers under the age of 18 each year.[7] Among adults who had ever smoked daily, 89% tried their first cigarette and 71% were daily users at or before age 18.[63] Among high school seniors who had used smokeless tobacco, 79% had first done so by the ninth grade.[63,215] By the time they are high school seniors, 22% of adolescents smoke daily.[216–218] Young people experiment with or begin regular use of tobacco for a variety of reasons related to social and parental norms, advertising, peer influence, parental smoking, weight control, and curiosity.[63,219] Nicotine dependence, however, is established rapidly even among adolescents.[220] Because of the importance of primary prevention in this population, clinicians should pay particular attention to delivering these messages to their patients. Specifically, because tobacco use often begins during preadolescence[221] clinicians should routinely assess and intervene with this population. Prevention strategies useful in more general settings can be found in the Institute of Medicine report *Growing Up Tobacco Free.*[222]

Young people vastly underestimate the addictiveness of nicotine. Of daily adolescent smokers who think that they will not smoke in 5 years, nearly 75% are still smoking 5–6 years later.[63] Of the nearly three fourths of adolescents (70.2%) who have ever tried smoking, more than one third (35.8%) became daily smokers during high school. Seventy percent of adolescent smokers wish they had never started smoking in the first place.[223] About three out of every four adolescent smokers have made at least one serious attempt to quit smoking and have failed.[224]

Tobacco Use Treatments in Children and Adolescents. A recent study has shown that adolescents' smoking status was identified in 72.4% of office visits, but smoking cessation counseling was provided at only 16.9% of clinic visits of adolescent smokers.[20] Therefore, clinicians both need to assess adolescent tobacco use and offer cessation counseling. Clinicians also should make an effort to prepare adolescents to quit smoking. For instance, clinicians may use motivational interventions such as those listed in Chapter 3B or consider techniques adapted for use with children.[56] Also, children and adolescents may benefit from community- and school-based intervention activities. The messages delivered by these programs should be reinforced by the clinician.[63]

A recent comprehensive review of adolescent cessation programs in a variety of settings has concluded that such programs produce quit rates that exceed naturally occurring quit rates, but that more and higher quality research needs to be done.[225]

Children and adolescents also benefit from the delivery to parents of information regarding second-hand smoke exposure. A review of the studies conducted by the expert panel showed that the delivery of information to parents regarding the harms of exposing children to second-hand smoke reduces childhood exposure to second-hand smoke and may reduce parental smoking rates.[162,226,227]

Because there is no evidence that bupropion SR or nicotine replacement is harmful for children and adolescents, clinicians may consider their use when tobacco dependence is obvious. However, because of the psychosocial and behavioral aspects of smoking in adolescents, clinicians should be confident of the patient's tobacco dependence and intention to quit before instituting pharmacotherapy. Factors such as degree of dependence, number of cigarettes per day, and body weight should be considered (see Table 4 for adult clinical recommendations).[228]

Future Research. The following topics regarding adolescents and children require additional research:

- The efficacy of advice and counseling.
- The efficacy of pharmacotherapy.
- The efficacy of interventions designed specifically to motivate youth to stop using tobacco.
- The efficacy of interventions designed to treat tobacco dependence in youth.
- The efficacy of child-focused versus family-focused interventions.
- The efficacy of treating parents' tobacco use in the context of pediatric visits.

Older Smokers

Recommendation: Smoking cessation treatments have been shown to be effective for older adults. Therefore, older smokers should be provided smoking cessation treatments shown to be effective in this guideline. (Strength of Evidence = A)

It is estimated that 13 million Americans ages 50 and older and 4.5 million adults over age 65 smoke cigarettes.[229] Smokers over the age of 65 can both quit smoking and benefit from abstinence.[16,230] Smoking cessation in older smokers can reduce the risk of myocardial infarction, death from coronary heart disease, and lung cancer. Moreover, abstinence can promote more rapid recovery from illnesses that are exacerbated by smoking and can improve cerebral circulation.[231,232] In fact, age does not appear to diminish the benefits of quitting smoking.[231]

The smoking cessation interventions that have been shown to be effective in the general population also have been shown to be effective with older smokers. Research has demonstrated the efficacy of the "4 A's" (ask, advise, assist, and arrange follow-up) in patients ages 50 and older.[233] Counseling interventions,[234–236] physician advice,[235] buddy support programs,[237] age-tailored self-help materials,[229,235,238] telephone counseling,[229,238] and the nicotine patch[239] have all been shown to be effective in treating tobacco use in adults ages 50 and older.

Because of particular concerns of this population (eg, mobility issues) the use of proactive telephone counseling appears particularly promising with older smokers.

Future Research. The following topics regarding older smokers require additional research:

- The efficacy of general tobacco use and dependence interventions, as well as those designed particularly for older smokers in promoting tobacco abstinence
- The efficacy of pharmacotherapy.
- Effective methods to motivate older smokers to make a quit attempt.

8. Special Topics

Background

Many additional factors that influence the efficacy of tobacco dependence interventions were considered by the panel. These include weight gain after cessation, the treatment of tobacco users other than cigarette smokers, and institutional barriers that may interfere with tobacco users receiving treatment. This chapter provides the panel's recommendations and supporting evidence on these disparate topics.

Weight Gain After Smoking Cessation

Recommendation: The clinician should acknowledge that quitting smoking is often followed by weight gain. Additionally, the clinician should: (1) note that the health risks of weight gain are small when compared to the risks of continued smoking, (2) recommend physical activities and a healthy diet to control weight, and (3) recommend that patients concentrate primarily on smoking cessation, not weight control, until ex-smokers are confident that they will not return to smoking. (Strength of Evidence = C)

Recommendation: For smokers who are greatly concerned about weight gain, it may be most appropriate to prescribe or recommend bupropion SR or NRT, in particular nicotine gum, which have been shown to delay weight gain after quitting. (Strength of Evidence = B)

The majority of smokers who quit smoking gain weight. Most will gain fewer than 10 pounds, but there is a broad range of weight gain, with as many as 10% of quitters gaining as much as 30 pounds.[240–242] However, weight gain that follows smoking cessation is a negligible health threat compared with the risks of continued smoking.[242,243]

Women tend to gain slightly more weight than men do.[242] For both sexes, African Americans, people under age 55, and heavy smokers (those smoking more than 25 cigarettes/d) are at elevated risk for major weight gain.[240,242,244–248]

For some smokers, especially women, concerns about weight or fears about weight gain are motivators to start smoking or continue smoking.[249–252] Adolescents, even as young as junior high age, who are concerned about their weight initiate smoking more often than do other adolescents.[253–256]

Concern about weight varies substantially by ethnicity. For example, adolescent African American females are much less likely to report that they smoke to control weight than are European Americans.[257] This is an important area for further study, as little tobacco research focuses on women of minority ethnicities.[257]

Some evidence suggests that attempts to prevent weight gain (eg, strict dieting) may undermine the attempt to quit smoking.[258–260] Other evidence suggests that weight gain is reduced if smoking cessation is accompanied by a moderate increase in physical activity.[261] One recent study showed that an exercise program, occurring in three 45-minute sessions per week, increases long-term smoking abstinence in women and delays weight gain when it is combined with a cognitive-behavioral smoking cessation program.[101]

Nicotine replacement—in particular, nicotine gum—appears to be effective in delaying postcessation weight gain. Moreover, there appears to be a dose-response relation between gum use and weight suppression (ie, the greater the gum use, the less weight gain occurs). However, once nicotine gum use ceases, the quitting smoker gains an amount of weight that is about the same as if she or he had never used gum.[246,262–265]

Bupropion SR also appears to be effective in delaying post-cessation weight gain.[118,266,267] However, once bupropion SR therapy is stopped, the quitting smoker, on average, gains an amount of weight that is about the same as if she or he had not used bupropion SR.[118,266]

Postcessation weight gain appears to be caused both by increased intake (eg, eating and alcohol consumption) and by metabolic adjustments. The involvement of metabolic mechanisms suggests that even if smokers do not increase their caloric intake upon quitting, they will, on average, gain some weight.[268–273] Once an individual relapses and

Table 46. Clinician Statements to Help a Patient Prepare For and Cope With Postcessation Weight Gain

Clinician statements
• "The great majority of smokers gain weight once they quit smoking. However, even without special attempts at dieting or exercise, weight gain is usually limited to 10 pounds."
• "There is evidence that smokers will gain weight once they quit smoking, even if they do not eat more. However, there are medications that will help you quit smoking and limit or delay weight gain. I can recommend one for you."
• "The amount of weight you will likely gain from quitting will be a minor health risk compared with the risks of continued smoking."
• "Try to put your concerns about weight on the back burner. You are most likely to be successful if you first try to quit smoking, and then later take steps to reduce your weight. Tackle one problem at a time! After you have quit smoking successfully, we can talk about how to reduce your weight."
• "I know weight is important to you, and that you don't want to gain a lot of weight. However, temporarily—just until you are confident that you have quit smoking for good—let's focus on strategies to get you healthy rather than on weight. Think about eating plenty of fruit and vegetables, getting regular exercise, getting enough sleep, and not eating a lot of fats. Right now, this is probably the best thing you can do for both your weight and your effort to quit smoking."
• "Although you may gain some weight after quitting smoking, compare the importance of this with the added years of healthy living you will gain, your better appearance (less wrinkled skin, whiter teeth, fresher breath), and good feelings about quitting."

begins smoking at precessation levels, he or she will usually lose some or all of the weight gained during the quit attempt.[272,274,275]

The research evidence reviewed above illustrates why concerns about weight gain are barriers to smoking abstinence. Many smokers (especially women) are concerned about their weight and fear that quitting will produce weight gain. Many also believe that they can do little to prevent postcessation weight except to return to smoking. These beliefs are especially difficult to address clinically because they are congruent with research findings; that is, the beliefs have some basis in fact.

Recommendations to Clinicians When Addressing Weight Gain. How should the clinician deal with concerns about weight gain? First, the clinician should neither deny the likelihood of weight gain nor minimize its significance to the patient. Rather, the clinician should inform the patient about the likelihood of weight gain and prepare the patient for its occurrence. However, the clinician should counter exaggerated fears about weight gain given the relatively moderate weight gain that typically occurs. Certain types of information may help prepare the patient for postcessation weight gain (see Table 46). Clinicians also should inform the patient that smoking presents a much greater health risk than the negligible health risk involved in the modest weight gain associated with smoking abstinence Second, before and during the quit attempt the clinician should stress that quitting smoking is the patient's primary, immediate priority, and that the patient will be most successful in the long run if he or she does not take strong measures (eg, strict dieting) to counteract weight gain during a quit attempt (see Table 46).

Third, during the quit attempt, the clinician should offer to help the patient address weight gain (either personally or via referral) once the patient has successfully quit smoking. Specifically, the clinician should recommend that intensive weight control strategies be avoided until the patient is no longer experiencing withdrawal symptoms and is confident that he or she will not return to smoking. The patient should, however, be encouraged to maintain or adopt a healthy lifestyle, including engaging in moderate exercise, eating plenty of fruits and vegetables, and limiting alcohol consumption.[101]

Future Research. The following topics regarding weight gain during tobacco dependence treatment require additional research:

- The impact of weight gain concerns on women of minority ethnicities.
- The efficacy of weight control measures during quit attempts and their effect on tobacco abstinence and weight.
- The efficacy of pharmacotherapy to control weight gain during quit attempts.
- The efficacy of the use of exercise to control weight gain during a quit attempt.
- The impact of weight gain concerns on adolescent smoking.

Noncigarette Tobacco Products

Recommendation: Smokeless/spit tobacco users should be identified, strongly urged to quit, and treated with the same counseling cessation interventions recommended for smokers. (Strength of Evidence = B)

Recommendation: Clinicians delivering dental health services should provide brief interventions to all smokeless/spit (chewing tobacco and snuff) tobacco users. (Strength of Evidence = A)

Recommendation: Users of cigars, pipes, and other noncigarette combustible forms of tobacco should be identified, strongly urged to quit, and offered the same counseling interventions recommended for smokers. (Strength of Evidence = C)

Like cigarette smoking, the use of smokeless or spit tobacco, such as chewing tobacco and snuff, produces addiction to nicotine and has serious health consequences. Consumption of such smokeless tobacco products has increased in recent years,[276–278] especially among young males. Health risks from these products include abrasion of teeth, gingival recession, periodontal bone loss, leukoplakia, and oral cancer and cardiovascular disease.[279–281] Thus, the use of smokeless tobacco is not a safe alternative to smoking.

Cigar smoking also poses serious health risks. Cigar smokers are at higher risk for coronary heart disease, COPD, lung and other cancers, with evidence of dose-response effects.[282,283] Cigar use is particularly concerning because cigar sales in the United States also have increased nearly 50% between 1993 and 1997.[282] In 1997, an estimated 6 million United States teenagers ages 14–19 (37% of males, 16% of females) smoked at least one cigar within the past year. Clinicians also should be aware of and address the use of other noncigarette tobacco products, including pipes, cigarillos, loose tobacco, bidis, and betel quid.

A close review of the literature showed that there is evidence that dental health clinicians (eg, dental hygienists) delivering brief advice to quit using smokeless/spit tobacco can increase abstinence rates.[284,285] Although somewhat limited, there is evidence that nonpharmacologic treatments used for smoking cessation also are effective in smokeless tobacco cessation. Therefore, clinicians should offer quitting advice and assistance to their patients who use tobacco regardless of the formulation of the tobacco product. Some information may be particularly relevant in the treatment of smokeless tobacco use. For instance, a large majority of moist snuff users have identifiable oral lesions, and this information may be useful in motivating a quit attempt.

Currently, there is insufficient evidence to suggest that the use of tobacco dependence pharmacotherapies increases long-term abstinence among users of smokeless tobacco. Specifically, studies conducted with nicotine gum and the nicotine patch have shown that these two medications have not increased abstinence rates in this population.[143]

Future Research. The following topics regarding noncigarette tobacco products require additional research:

- The efficacy of advice and counseling treatments to promote abstinence among users of noncigarette tobacco products.
- The efficacy of pharmacotherapy treatments to promote abstinence among users of noncigarette tobacco products.
- The efficacy of combined pharmacotherapy and counseling and behavioral therapies with users of noncigarette tobacco products.

Clinician Training

Recommendation: All clinicians and clinicians-in-training should be trained in effective strategies to assist tobacco users willing to make a quit attempt and to motivate those unwilling to quit at this time. (Strength of Evidence = B)

The above recommendation is based on panel review of the published literature rather than a formal meta-analysis. Relevant studies focused primarily on physician training. Many of these studies examined the impact of training as it co-occurred with other systems changes such as reminder systems or staff education.[286,287] Training appears to be more effective when coupled with these systems changes.

Clinicians must be trained in effective tobacco use treatments if guideline recommendations are to be implemented. The importance of training is clear in that clinicians report lack of relevant knowledge as a significant barrier to intervening with their patients who use tobacco.[288–291]

Training should be directed at both clinicians-in-training as well as practicing clinicians. Training should be reinforced throughout the clinicians' education and practice.[292] For clinicians-in-training, most disciplines neither provide training nor require competency in tobacco use interventions. For example, an NCI expert panel found that medical schools do not consistently train students in effective smoking cessation interventions.[293] That panel recommended that a specific curriculum devoted to tobacco dependence treatment be included as part of each medical student's education. This curriculum may be taught as part of a preventive medicine or substance abuse course or as a class by itself. Similar recommendations would be relevant to virtually all other clinical disciplines. More recently, a survey of United States medical schools found that most medical schools (69%) did not require clinical training in tobacco dependence treatment.[289] Training in tobacco use interventions should not only transmit essential treatment skills but also inculcate the belief that cessation treatment is a standard of good practice.[294]

Practicing clinicians also would benefit from continuing education that addresses tobacco dependence treatment. This guideline recommends that clinicians be reimbursed for treating tobacco dependence and that their intervention activities be tracked. Either of these policies should foster increased interest in establishing expertise in treating tobacco use among practicing clinicians.

Several factors would promote the training of clinicians in tobacco intervention activities:[293]

- Inclusion of education and training in tobacco dependence treatments in the required curricula of all clinical disciplines.
- Inclusion of questions on effective tobacco dependence treatment in licensing and certification exams for all clinical disciplines.
- Adoption by specialty societies of a uniform standard of competence in tobacco dependence treatment for all members.

Finally, clinicians who currently use any tobacco product should participate in treatment programs to stop their own tobacco use permanently. Clinicians have an important role as models for their patients. Therefore, it is heartening that many types of clinicians have dramatically decreased their tobacco use over the past 20 years. In a report on tobacco-use prevalence by occupation, the rate of smoking was noted to be 5.5% among physicians, 7.4% among dentists, 8.7% among physical therapists, and 22.0% among registered nurses.[295] All of these prevalence rates are lower than tobacco-use rates in the general population.

Future Research. The following topics regarding clinician training require additional research:

- The efficacy of training programs for other health disciplines such as nursing, psychology, dentistry (including hygienists), social work, and pharmacy.
- The effective elements in successful training programs (eg, continuing medical education, interactive components).
- The efficacy of systems changes, such as reminder systems (and performance feedback), when implemented with training programs.

Economic Aspects of Tobacco Dependence Treatments and Health Systems Interventions

Recommendation: The smoking cessation treatments shown to be efficacious in this guideline (both pharmacotherapy and counseling) are highly cost-effective relative to other reimbursed treatments (eg, treatment of hyperlipidemia and mammography screening) and should be provided to all smokers. (Strength of Evidence = A)

Recommendation: Intensive smoking cessation interventions are especially efficacious and cost-effective, and smokers should have ready access to these services as well as to less intensive interventions. (Strength of Evidence = B)

Recommendation: Smoking cessation treatments (both pharmacotherapy and counseling) should be included as a paid or covered benefit by health benefits plans because doing so improves utilization and overall abstinence rates. (Strength of Evidence = B)

Recommendation: Sufficient resources should be allocated for clinician reimbursement and systems support to ensure the delivery of efficacious tobacco use treatments. (Strength of Evidence = C)

Recommendation: Provision of guideline-based interventions to treat tobacco use and addiction should be included in standard ratings and measures of overall health care quality (eg, National Committee for Quality Assurance's Health Plan Employer Data and Information Set [NCQA HEDIS], the Foundation for Accountability [FACCT]). (Strength of Evidence = C)

Smoking cessation treatments ranging from brief clinician advice to specialist-delivered intensive programs, including pharmacotherapy, are not only clinically effective, but also they are extremely cost-effective relative to other commonly used disease prevention interventions and medical treatments. Cost-effectiveness analyses have shown that smoking cessation treatment compares quite favorably with routinely reimbursed medical interventions such as the treatment of hypertension and hypercholesterolemia, as well as with preventive screening interventions such as periodic mammography or Papanicolaou smears.[28,71,75–77,296–298] Treating tobacco dependence is particularly important economically in that it can prevent a variety of costly chronic diseases, including heart disease, cancer, and pulmonary disease. In fact, smoking cessation treatment has been referred to as the "gold standard" of preventive interventions.[78]

It is important to note that smoking cessation is also cost-effective in special populations such as hospitalized patients and pregnant women. For hospitalized patients, successful tobacco abstinence not only reduces general medical costs in the short-term, but also reduces the number of future hospitalizations.[16] Smoking cessation interventions for pregnant women are especially cost-effective because they result in fewer low-birthweight babies and perinatal deaths, fewer physical, cognitive, and behavioral problems during infancy and childhood, and also yield important health benefits for the mother.[299,300]

Although data suggest that, among clinical interventions, intensive interventions are more cost-effective than low-intensity interventions,[64] widely disseminated public health interventions may have even greater cost-effectiveness.[301]

The failure of a health plan to cover tobacco dependence treatment as an insured benefit could reduce access to these services and reduce the number of people seeking these services. It has been found that when smoking cessation services are provided as a fully covered benefit by a health plan in contrast to a health plan that required a significant co-pay, the overall utilization of cessation treatment increases and smoking prevalence within the health plan will decrease.[66] Moreover, the presence of prepaid or discounted prescription drug benefits increases patients' receipt of pharmacotherapy and smoking abstinence rates.[136,302,303]

Primary care clinicians frequently cite insufficient insurance reimbursement as a barrier to the provision of preventive services such as smoking cessation treatment.[304] An 8-year insurance industry study found that reimbursing physicians for provision of preventive care resulted in reported increases in exercise, seat belt use, and weight loss, as well as decreased alcohol use and a trend toward decreased smoking.[305]

It may be in the best interests of insurance companies, managed care organizations, and governmental bodies within a specific geographic area to work collectively to ensure that tobacco dependence interventions are a covered benefit. This also would allow the financial benefits of these services to be realized by all the health plans within a community.

The provision of tobacco dependence treatment should be increased by: (1) attention to health plan "report cards" (eg, NCQA HEDIS),[306,307] which support smoker identification and treatment, and (2) accreditation criteria used by the Joint Commission on Accreditation of Health Care Organizations and other accrediting bodies, which include the presence of effective tobacco assessment and intervention policies.

Future Research. The following topics regarding cost-effectiveness and health systems require additional research:

- To what extent are the various tobacco dependence treatments cost-effective, both short and long term?
- What is the best way to remove systemic barriers that prevent clinicians from effectively delivering tobacco dependence treatments?
- What are the best systemic interventions to encourage provider and patient utilization of effective tobacco dependence treatments?
- Whether reimbursement for tobacco dependence treatment is recovered later in reduced health care costs.
- Evaluation of the relative costs and economic impacts of different formats of efficacious treatments (eg, proactive telephone counseling, face-to-face contact, pharmacotherapy).

Alternative Treatment Goals: Harm Reduction

There is insufficient evidence to support a recommendation regarding harm reduction interventions. In harm reduction strategies, tobacco users alter, rather than eliminate, their use of nicotine or tobacco to reduce or avoid its harmful consequences. Many harm reduction strategies have been proposed such as reduced use of tobacco (perhaps with the conjoint use of pharmacotherapy), use of less hazardous tobacco/nicotine products, or use of less addictive tobacco/nicotine products.[308] It is difficult to evaluate the potential benefits of harm reduction strategies because of a lack of published data. For instance, it is unknown whether public health would ultimately be better served by smokers' attempting to reduce or shift their tobacco use (eg, smoke fewer cigarettes) rather than by making repeated quit attempts. It must be borne in mind that all types of tobacco use (eg, smokeless, pipe, cigar) carry significant health risks. Specifically, the use of smokeless/spit tobacco is not a safe alternative to smoking. Moreover, increased health risks have been documented even in "light" smokers (less than five cigarettes per day).[309] Finally, evidence suggests that when smokers are forced to reduce their nicotine intake, they frequently engage in compensatory smoking (eg, taking more puffs per cigarette, taking deeper puffs).[310]

In sum, it is not known whether harm reduction strategies would reduce tobacco exposure over the long-term, whether they would reduce negative health outcomes, whether their encouragement would yield greater benefits than an exclusive reliance on abstinence, and whether they might increase tobacco use prevalence by suggesting the availability of a "safe" tobacco strategy. As a result, prior to embracing any harm reduction strategy, extensive research will need to be performed on the feasibility, efficacy, and costs and benefits of these strategies. Some of the needed research includes:

- Are a significant number of smokers able to maintain smoking reduction behaviors (eg, reduced quantity) for long periods of time? What techniques promote such reductions, and what are the characteristics of those who are able to reduce and those who are unable to do so?
- What degree of compensatory smoking occurs when smokers reduce the number of cigarettes smoked or smoke low tar cigarettes when:
  - –Aided by medications (eg, bupropion SR or NRT)
  - –Unaided by medications
- What are the physiologic and health impacts of smoking reduction strategies (ie, reduced smoking rate or use of low tar and/or low nicotine cigarettes) when:
  - –Aided by medications (eg, bupropion SR or NRT)
  - –Unaided by medications
- Will a significant number of smokers use nontobacco medicines (eg, bupropion SR and/or NRT) in lieu of smoking over the long term, and will this benefit health?
- What are the public health and clinical implications of recommending a harm reduction strategy in addition to abstinence strategies (eg, a message such as "You should quit. If you can't quit, you should try to reduce.")?
- Would the forced reduction of the nicotine content of cigarettes by the tobacco companies constitute an effective societal intervention?[311]
- What medications are most effective in promoting reduced smoking (short and long term) without compensatory smoking?
- How effective are behavioral interventions in promoting reduced smoking (short and long term) without compensatory smoking?

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Unusual Ventilator Graphics Encountered Following Successful Resuscitation from Cardiac Arrest

Jeffrey M Haynes RRT RPFT

A 63-year-old white male patient with a history of hypertension, dyslipidemia, and tobacco abuse presented to the emergency department following cardiac arrest. Out-of-hospital resuscitation had successfully converted ventricular fibrillation to sinus tachycardia with intravenous epinephrine and defibrillation. Upon arrival to the emergency department the patient was unconscious and hypotensive. The patient had been orally intubated in the field and was having spontaneous respirations augmented with high-flow oxygen via a bag-valve ventilator. Auscultation of the chest revealed equal hemithoracic ventilation and diffuse panrespiratory rhonchi and coarse crackles. An anteroposterior chest radiograph showed appropriate endotracheal tube positioning, florid pulmonary edema, and bilateral hydrothoraces. A 12-lead electrocardiogram suggested acute anterior wall myocardial injury. The patient was placed on a Puritan-Bennett 7200 ventilator in the assist control mode, with the respiratory rate set at 16 breaths per minute, the tidal volume (V_T) at 800 mL, and the fraction of inspired oxygen at 1.0. The total respiratory rate was 28 breaths/minute. Arterial blood gas analysis indicated pH 7.26, arterial partial pressure of carbon dioxide 36 mm Hg, arterial partial pressure oxygen 98 mm Hg, and arterial oxygen saturation 0.96. Positive end-expiratory pressure (PEEP) of 5 cm H_2O was added to the ventilator settings and the patient was later transferred to the intensive care unit with acute myocardial infarction complicated by cardiac arrest, lactic acidosis, hydrostatic pulmonary edema, and cardiogenic shock. Figure 1 shows the ventilation graphics encountered by the respiratory therapist in the intensive care unit.

What ventilation abnormalities are illustrated by the pressure-time and flow-time ventilator graphics in Figure 1?

The pressure-time graphic shows slow decay of airway pressure from the peak inspiratory pressure (PIP) to applied PEEP (PEEPa). The areas labeled "1" on the pressure-time graphic in Figure 2 represent airway pressures greater than PEEPa during the expiratory phases. The flow-time graphic shows low expiratory flows that do not appear to reach zero before the next inspiratory phase. These slow expiratory flow signals, labeled "4" on the flow-time graphic in Figure 2, do not decay in an exponential fashion and suggest significant resistance to expiratory air flow. In short, the graphics in Figure 1 appear to depict inadvertent expiratory retard.

What do you suspect is the cause of the ventilation abnormalities in this case?

Suspicion should be directed at the expiratory side of the ventilator circuit, since the abnormality occurs while gas flows exclusively in that direction. Obviously expiratory flow can be compromised by obstructions within the patient's airways or endotracheal tube, but the back pressure generated by an obstruction at these sites would not be detected by a pressure manometer proximal to the endotracheal tube, as depicted in Figure 1. In other words, if the source of increased flow resistance is upstream from the circuit Y-piece, end-inspiratory pressure within the ventilator circuit should still rapidly decay once the circuit is exposed to the atmosphere by the exhalation valve.[1] Therefore, examination of the pressure-time graphic may yield information about the site of an expiratory obstruction. An additional method that may isolate an obstruction within the expiratory conduit is to disconnect the expiratory tubing from the ventilator or the circuit Y-piece at the outset of exhalation and then gauge whether expiratory flow out of the chest increases. The development of inadvertent expiratory retard in this case was caused by an obstruction within the exhalation compartment of the ventilator. The ventilator was equipped with an exhalation compartment design utilizing a frontal access door. This design requires some manual assembly of the exhalation compartment components. Specifically, a section of tubing (check valve hose) with a short elbow on its proximal end

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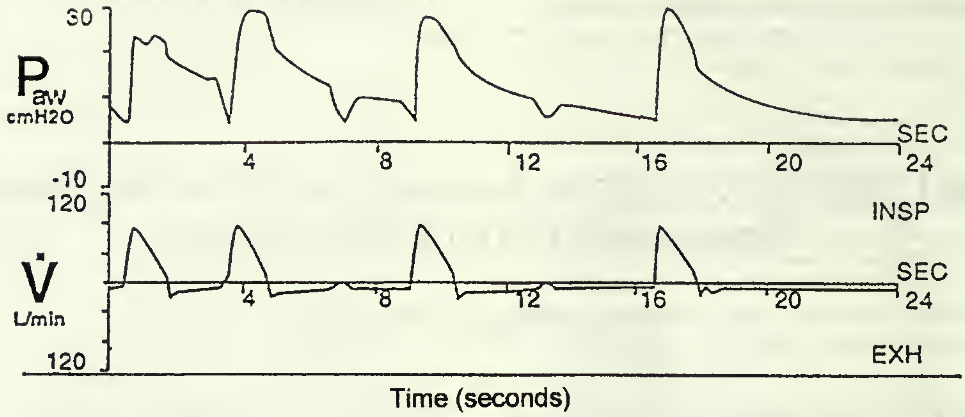

Fig. 1. Pressure-time (upper) and flow-time (lower) ventilator graphics recorded from a patient following successful resuscitation from cardiac arrest.

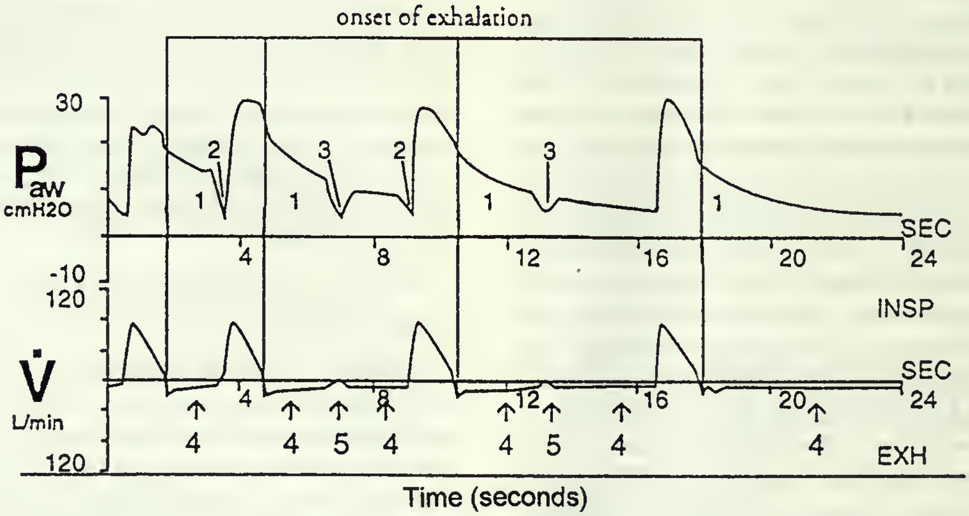

Fig. 2. Analysis of the ventilation abnormalities. Areas labeled 1 represent prolonged elevation of airway pressure. Drops in airway pressure labeled 2 depict excessive inspiratory efforts required to trigger the ventilator. Drops in airway pressure labeled 3 and expiratory flow labeled 5 represent inspiratory efforts made by the patient without ventilator responses. Expiratory flow patterns labeled 4 reveal reduced expiratory flow.

needs to fastened to the outlet of the bacteria filter leaving the expiratory side of the ventilator circuit. This tubing serves as a conduit for gas to travel from the bacteria filter to the exhalation valve. Inspection of the exhalation compartment following the discovery of expiratory retard revealed that the check valve hose had been attached to the bacteria filter outlet in a position counterclockwise to the manufacturer's recommended position. This incorrect positioning produced a kink in the pliable check valve hose, thus impeding gas flow to the exhalation valve and causing the expiratory retard. Repositioning of the check valve hose relieved the expiratory retard, as shown by Figure 3. This exhalation compartment design has been reengineered by the manufacturer, which has eliminated the need for this assembly procedure.

Is there evidence in Figure 1 that inadvertent PEEP (PEEPi) may be present?

Yes. The flow-time graphic in Figure 1 appears to show expiratory flows not reaching zero, which implies PEEPi from incomplete exhalation (ie, gas trapping). In addition, although definitive PEEPi measurements were not made, the fact that breaths in Figure 1 appear to begin at expiratory pressures higher than PEEPa (an unusual finding when the expiratory valve is presumed to be opened to the atmosphere) also implies the existence of upstream PEEPi. In fact, the discovery of end-expiratory pressures greater than zero or PEEPa is precisely the criterion that is used to define PEEPi when measured by the end-expiratory plateau maneuver.[1]

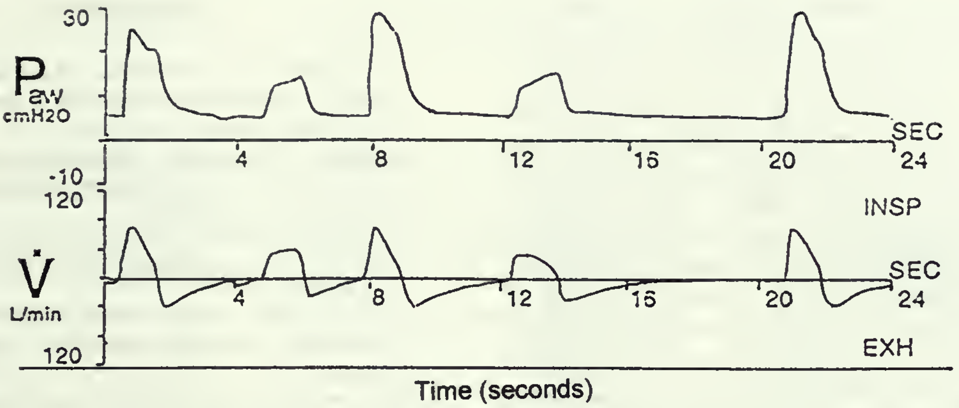

Fig. 3. Pressure-time and flow-time graphics recorded shortly after the removal of the expiratory compartment obstruction. Note that the patient had been changed to synchronized intermittent mandatory ventilation and pressure support.

Is there evidence in Figure 1 that the patient's minute ventilation may have been compromised?

Yes. In Figure 2, the drops in airway pressure (labeled 3) and expiratory flow (labeled "5") indicate patient inspiratory efforts that failed to trigger a volume control V_T. The expiratory retard and apparent PEEPi has widened the pressure gradient between the native airway and PEEPa, requiring greater reduction in airway pressure to signal the ventilator to begin insufflation of an assisted V_T. This patient-ventilator dyssynchrony may have been an impediment to more complete respiratory compensation of metabolic acidosis.

Does Figure 1 suggest an increase in the work of breathing (WOB) imposed by the ventilator?

Yes. The presence of expiratory retard and apparent PEEPi appears to have widened the pressure gradient between the native airway and PEEPa. The ventilator was programmed to utilize pressure-triggering with a sensitivity of –1 cm H_2O, requiring the patient to reduce airway pressure to 4 cm H_2O (ie, 1 cm H_2O less than the PEEPa of 5 cm H_2O) to trigger the delivery of a volume control V_T. In Figure 2 the inspiratory efforts successfully triggering volume control V_T (labeled "2") and the unsuccessful inspiratory efforts (labeled "3") demonstrate the patient producing reductions in airway pressure of up to 10 cm H_2O in an attempt to satisfy his ventilatory drive. These increased inspiratory efforts necessitate greater changes in transpulmonary pressure and, thus, greater WOB. Setting PEEPa to just below PEEPi can effectively curtail this added inspiratory threshold load.[2–4] In addition, the presence of expiratory retard suggests increased expiratory resistance, which may also impose increased flow-resistive WOB[5] and worsen dyspnea[6] when exhalation is not passive. It is important to keep in mind that when one speaks of work of breathing, the term *work* implies that a change in volume occurs as a result of the pressure applied; if volume change does not occur from a change in pressure, the term "oxygen cost of breathing" may be more accurate in that it signifies total respiratory muscle energy expenditure.

Discussion

Expiratory retard is a ventilatory maneuver available on earlier generations of mechanical ventilators. Application of expiratory retard impedes expiratory flow, which slows the decay of airway pressure. This appears on the expiratory pressure tracing as a more gradually declining pressure, as opposed to the exponential pressure decay seen when expiratory retard is not imposed on the ventilator circuit. The rationale for the use of this technique is to improve bronchoalveolar emptying in patients with severe air flow obstruction by counteracting the tendency of extraluminal pressure to produce dynamic airways collapse and subsequent hyperinflation.[7] This technique is not commonly employed during mechanical ventilation anymore, but is probably the primary mechanism that ameliorates dyspnea and air trapping in emphysema patients during pursed-lip breathing.[8] In this case, however, dynamic hyperinflation appears to have been a consequence of inadvertent expiratory retard related to elevated exogenous expiratory resistance. Definitive detection and quantification of PEEPi can be accomplished by the utilization of an esophageal balloon[9] or by measurement of end-expiratory plateau pressure,[1] but PEEPi levels can be quite dynamic.[9] PEEPi is very common among ventilated patients[2,9–12] and can have important consequences, including risk of volutrauma, altered hemodynamics, and increased WOB.[1–4] It is important to realize that PEEPi is not exclusively a product of a patient's pathophysiology; it can also be affected by the expiratory conduit we impose and the expiratory time we allow.[12–14] This case provides a good illustration that not all PEEPi is intrinsic. In the same way, WOB is also not only a product of a patient's pathophysiology and metabolic demands: it is also affected by artificial airways and mechanical ventilators. Despite the perception that we help patients with ventilatory insufficiency in part by alleviating their WOB with mechanical ventilators (and we do), it is critical to keep in mind that our artificial airways and sophisticated ventilatory apparatus also impose work.[5,15–17] This imposed work is often derived from additional inspiratory and expiratory resistance and the inherent inferiority of gas availability to the patient as compared to breathing directly from the atmosphere. It is therefore incumbent on clinicians to consider the work they impose on the patient during the process of assisted ventilation, from the moment the patient is intubated until extubation. In this case the imposed WOB appears to have been accentuated by a ventilator malfunction related to a circuit assembly error.

The widespread availability of ventilator graphics is an exciting development that can aid clinicians in assessing ventilation in terms of the volume, pressure, and flow traces. In this case ventilator graphics were helpful in the detection and evaluation of this ventilator malfunction. However, despite the usefulness of ventilation graphics in this case, they should not be considered the only means for identifying this or many other ventilation abnormalities. The expiratory retard in this case was also visible on the ventilator's pressure manometer and, more importantly, was quite obvious on physical exam. Simple inspection of the patient revealed impaired expiratory thoracic deflation and inspiratory efforts being made by the patient without ventilator responses. The practice of performing physical examination to detect ventilation abnormalities is not just some prim ritual; recent data suggest that physical examination can be useful for the detection of PEEPi,[18] which might be an important skill to have when ventilator graphics are not available (eg, ventilating with a bag-valve ventilator). The principal benefit of ventilation graphics in this case was perhaps its striking visual display of the abnormality, which was recognized by the intensive care unit respiratory therapist immediately upon entering the patient's room, which expedited the correction of the malfunction. Ventilator graphics can provide clinicians with valuable real-time information that should be considered as an adjunctive source of data, not a replacement for other components of a comprehensive patient evaluation.

ACKNOWLEDGMENTS

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Cellular and Molecular Biology of Nitric Oxide. Jeffrey D Laskin and Debra L Laskin, editors. New York: Marcel Dekker. 1999. Hardcover, illustrated, 388 pages, $185.

In the last few years there has been an overwhelming quantity of literature on nitric oxide (NO), a small but highly reactive molecule. This abundant literature reflects the great interest in this important biological mediator substance among scientists from several areas, including biochemistry, cellular and molecular biology, and experimental medicine. Unlike the increasing development in basic science, however, the clinical application of gaseous NO as a new, encouraging approach to treat lung diseases in adults with acute or chronic pulmonary hypertension currently lacks much interest, at least in Europe, because of several disappointing therapeutic studies. Hence, as a clinician, one occasionally wonders: it is actually worthwhile to read new articles or books about NO? That it *is* in fact worthwhile is impressively proven by Jeffrey and Debra Laskin in their book **Cellular and Molecular Biology of Nitric Oxide**, a reference that discusses the latest advances in NO research for molecular and cell biologists, biochemists, pharmacologists, toxicologists, physicians, respiratory physiologists, and graduate and medical students in these disciplines. Nurses and therapists caring for clients with respiratory problems, even those using inhaled NO in the intensive care unit, are obviously not the intended audience of this book, since aspects of exogenous, administered NO in pulmonary diseases are not taken into account.

Indeed, based on the table of contents, the reader might assume that this book refers exclusively to theoretical topics about NO, with little or no relevance for clinical use or diagnostic approaches. Not so. The book, comprising almost 400 pages, focuses on the convergence of new technologies such as genomics and genetic engineering, via regulatory biochemistry and cellular biology, with clinical questions in pharmacology, pathophysiology, and new therapies. A common feature of all 15 chapters is the special effort of the 34 contributors (all world experts in the NO field) to concentrate on the latest advances. Chapters 1–3, 7, and 12 discuss new findings on NO synthesis by transcriptional regulation, arginine metabolism, and activity of the NO synthase isoforms. The first two chapters in particular offer a detailed description of NO synthesis regulation, together citing more than 400 references and thus giving specialists in this field the opportunity to consult up-to-date original papers in many first-class journals. Chapter 4 deals with interactions of oxygen-dependent radicals and NO on the biochemical level. In this context, the relevance of NO to carbon monoxide intoxications, definitely an exciting new field in toxicology, is discussed. Chapter 5 encloses the broad field of NO synthase inhibitors, which are not only a pivotal tool in studying cellular effects of NO, but are also currently evaluated as a new clinical approach for therapy in diseases with increased local and/or systemic NO synthesis. Interestingly, the authors make it obvious that the so-called "specificity" of different NO synthase inhibitors for the described enzyme subtypes is limited—an important fact that should be considered in interpreting experimental and clinical studies. The dual role of NO in the pathogenesis of tissue injury is described in Chapter 9: the authors explain how NO modified tissue injury in several experimental models, either protecting the cells or increasing the pathologic damage, depending on the toxicant, the target tissue, and the extent to which toxicity is mediated by reactive oxygen species. A major part of this chapter is the description of the regulatory role of NO in the pathogenesis of lung injuries due to specific effects on alveolar cells and pulmonary macrophages.

This modulating effect of NO during inflammation processes is also stressed in other sections of the book. Chapters 8 and 10 describe the central role of NO, reactive oxygen species, and peroxynitrite during inflammatory diseases. In the broad field of autoimmune disorders such as rheumatoid arthritis, glomerulonephritis, and vasculitis, the interaction of these radicals contribute to the pathology. Mononuclear cells and chondrocytes can produce NO via NO synthase action, and regulation of this enzymatic activity by several cytokines results in proinflammatory or antimicrobial effects. The pharmacology of some antioxidant drugs such as 5-aminosalicylic acid, which interfere with NO effects, is described, and the heterogeneity and complexity of NO effects in the pathogenesis of these diseases are highlighted. Chapters 11 and 15 address the programmed cell death (apoptosis) induced by NO. This important and highly regulated mechanism used for the clearance of damaged cells is induced by several transduction pathways that may include NO as a crucial mediator. Whereas apoptosis basically seems to be an autoregulatory process within injured cells, NO synthesis seems to be a way by which the start signal for apoptosis is transferred between cells because of the low molecular weight and lipophilic nature of NO—overall an important mechanism by which NO mediates cellular toxicity.

Other topics include some of the organ-specific NO effects. Chapter 6 deals with homeostatic and pathologic actions of NO in the kidney. The basic physiology of renal function is briefly reviewed, and the influence of NO on the renin-angiotensin system is pointed out. NO can stimulate or inhibit renin release, probably depending on the vascular shear stress. The chapter also covers the contribution of NO in renal ischemia/reperfusion injury and the role of osteopontin as an important phosphoprotein that ameliorates NO-induced injury by virtue of its ability to inhibit induction of NO synthase activation. Chapter 15 describes NO's specific role in regulating apoptosis and necrosis in hepatic cells. The authors emphasize that increasing knowledge obtained via further dissection of NO-dependent signaling pathways involved in either necrosis or apoptosis may lead to clinically applicable interventions. The functions and consequences of NO production in the skin is another organ-specific role of NO described in Chapter 12. It is pointed out that NO is integral to virtually every aspect of skin physiology, regulating keratinocyte proliferation, differentiation, activation, and cell death—"a challenge for the future," the authors stress. Finally, some aspects of new interventional strategies are addressed in Chapters 13 and 14. Taurine chloramine, an inhibitor of the inducible NO synthase expression, is introduced as a potential modulator of inflammation by inhibiting NO synthesis in activated macrophages. But also many natural products such as tea, rosemary, curcumin, capsaicin, genistein, and omega-3 polyunsaturated fatty acids can inhibit NO synthesis and thereby act as anti-inflammatory agents. The authors conclude that natural products provide a reservoir of different substances that may act as modulators of the NO pathway.

Undoubtedly, it must have been very difficult for the editors to put this overwhelming quantity of information in a uniform format, but I think that they have achieved their aims. The overall appearance of the book is very good, although some of the contributions differ, which, considering the number of contributors, is not surprising. Chapter 4, for example, is presented like an original paper, which might help to follow the lab methods, but, on the other hand, limits the possibility to offer a didactic survey. In most cases, however, the chapters are well structured, beginning with an introduction to the topic, often accompanied by a historical review. The different aspects of the topics are put into titled paragraphs, each of which can be read as its own section, providing clear and logically sound findings and arguments. The writing style is mostly clear and concise, and the number of typographical errors is very low. The overall quality of the figures and tables is excellent, occasionally disturbed by low-quality computer scans or photograph reprints (eg, on pages 138 and 173), but frequently enriched by clear and ingenious schemes and cartoons. The references of each chapter are useful and thorough. The arrangement of the chapters sometimes seems confusing. For example, regulation of NO synthesis is covered in Chapters 1–3, 5, 7, and 12–14, NO's effects on programmed cell death are covered in Chapters 11 and 15, NO's role in inflammation is discussed in Chapters 8–10, and 13, and organ-specific NO effects are discussed in Chapters 6, 12, and 15. This overlap is probably because of the overlap between many aspects of NO research, and the options for an alternative arrangement were limited. Unfortunately, the index is not well structured and is probably the weakest part of the book. The head words are without any subspecialties, and referring pages are simply listed, probably by an automated computer search. What does it help to enclose the word "death" with 22, "rat" with 43, or "human" with 44 page numbers without any subheadings? This deficiency should be corrected in the next edition.

Hence, **Cellular and Molecular Biology of Nitric Oxide** is not an encyclopedia, ie, "a work containing exhaustive information on some one branch of knowledge" (Oxford English Dictionary). However, I am optimistic that the book will reach a broad readership among physicians and scientists, although the interest of therapists and nurses will probably be limited. Taking into account the current status of inhaled NO (ie, still an experimental approach, at least in adult pulmonary medicine), it will be several more years until NO is accepted as a daily therapeutic agent. For the basic science researcher, this extraordinarily detailed book gives a broad and up-to-date view of developments in NO research, which helps researchers to keep in touch with each other as well as with the clinicians who are testing NO as a therapy for several diseases. For the clinician, although some of the specialized chapters might be difficult to read, the book offers a view into the future, thus encouraging researchers to continue clinical evaluation of NO application as a fascinating opportunity to influence cell biology, but also reminding us that NO is a pleiotropic mediator of human physiology, and not only a short-living inhaled drug. In the preface, the editors express their hope "that the book will further fuel the interest in the rapidly expanding field of NO biology and encourage additional research into understanding the role of NO in the pathogenesis of disease and the development of therapeutic agents that will be useful in human medicine." I feel that this is not only a justified hope, but, based on the presented result, a fact for which both the editors and contributors are to be congratulated. **Cellular and Molecular Biology of Nitric Oxide** is a fascinating tool that helps us to not lose reference to cellular mechanisms of endogenous and exogenous NO in clinical use. I learned a lot.

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Physiology of Sport and Exercise, 2nd edition. Jack H Wilmore and David L Costill. Champaign, Illinois: Human Kinetics. 1999. Hardcover, illustrated, 710 pages, $59.

Exercise physiology is the study of how the structures and functions of the body are altered during the stresses of acute and chronic exercise training. Sport physiology extends the concepts of exercise physiology to athletic endeavor. In **Physiology of Sport and Exercise**, Wilmore and Costill provide a basic yet thorough introduction to exercise and sport physiology for students preparing for careers in this area. The book also serves as a basic resource for health professionals who work with athletes or patients in exercise rehabilitation programs.

According to the authors, the key question answered in this book is "How does your body respond to the high physiologic demands of physical activity?" And their primary goal is "to build upon the foundation of knowledge that you have constructed through basic course work in human anatomy and physiology by applying the principles you've learned to how the body performs and responds to physical activity." In my opinion, the authors have answered this question and met their goal. Wilmore and Costill are two of the most respected names in exercise and sport physiology, and each chapter captures their authoritative overview of the critical issues.

The book is divided into 7 parts and 21 chapters. Parts I through III review selected physiologic systems, focusing on their responses to acute and chronic exercise. This review includes muscular and neurological control of movement, metabolism and basic energy systems, hormonal regulation of exercise, and cardiovascular and respiratory regulation and adaptation. Part IV covers environmental influences on performance, including heat, cold, altitude, and the weightlessness of space. Part V focuses on issues related to athletic performance, including nutrition, ergogenic aids, training systems, overtraining, and body composition. Part VI examines the influences of growth and development, old age, and gender on sports performance. Finally, Part VII gives a brief overview of the relationship between physical activity and cardiovascular disease, obesity, and diabetes.

I felt that each part of the book, except for VII, was strong, and at least as good as competing texts. Part VII was definitely the weakest of the book, especially the section on diabetes, which failed to incorporate recent findings. The topic of physical activity and health is obviously not a focus of this textbook, but I felt the authors could still have provided a stronger review in this burgeoning area of sports medicine. I was also disappointed with the chapter on ergogenic aids and performance, because the authors failed to provide sufficient information on ginseng, mahuang, inosine, boron, creatine, and other substances of current interest. The authors are well known for their research in sports nutrition, thermoregulation, body composition, metabolism and energetics, hormonal regulation, muscle fiber typing, chronic adaptations to exercise training, aging, gender influences on performance, and sports performance and testing, and these sections of the book were especially good. Wilmore and Costill also ventured down new roads in exercise physiology, including exercise immunology, exercising in space, and exercising underwater. Their introductory chapter had an interesting historical perspective on exercise and sport physiology.

The book is designed for the student reader with the goal of making learning easy and enjoyable. To this end, the book features several pedagogical aids, including: each part of the book is color-coded; chapters begin with a brief overview and outline; key points in each chapter are placed in boxes for quick reference; key terms are highlighted in red within the text; and review boxes provide concise summaries throughout each chapter. The book is supplemented with strong and appropriate references and selected readings, study questions, and a comprehensive glossary and index. I found each of these aids to be very useful as I reviewed the book.

The text is supported by numerous figures, photos, and tables, most of them beautifully crafted and illustrative of basic principles. Each figure and table is supported with a scientific reference, many of them based on the excellent work of the authors. The book is attractive and colorful, and is not too cluttered with boxes and illustrations. The writing style is clear, concise, and highly readable, and I am sure that upper-division college students will find the book to be exactly at their level.

I highly recommend this book both as a textbook for exercise science majors and as a reference text for all health professionals. The extensive research and academic backgrounds of the two authors shine through in each chapter, and no other competing text can claim such a respected authorship. The second edition was published five years after the first, which has been strengthened and improved. The publishers have produced a highly attractive text rich in learning aids and illustrations, while the authors have ensured that the text includes all of the important content in exercise and sport physiology.

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Management of Respiratory Tract Infections, 2nd edition. John G Bartlett MD. Philadelphia: Lippincott Williams & Wilkins. 1999. Softcover, 275 pages, $14.95.

Respiratory tract infections exact an enormous cost on humanity in terms of morbidity and mortality, as well as economic burden. Lower respiratory tract infection remains the leading cause of death from infectious disease both in the United States and globally. Though associated with less mortality, upper respiratory infection is one of the most common reasons for patients to seek care and are a source of substantial economic burden because of missed work and pharmaceutical expenditure. Furthermore, while the last decades of the 20th Century provided practitioners with a markedly increased antibiotic armamentarium, emerging resistance among respiratory pathogens poses an increasing concern and necessitates appropriate antibiotic use. The second edition of **Management of Respiratory Tract Infections** seeks to provide a concise, clinically oriented guide to conditions associated with infection of this organ system. In a series of well-organized chapters, this compact paperback book reviews epidemiology, diagnostic methods, management decisions, and antibiotic use in pneumonia, cough syndromes, the common cold, streptococcal pharyngitis, and sinusitis. Information is presented as a combination of carefully referenced text and tables that either highlight important clinical points or organize detailed information. Each major section of the book is preceded by a useful "snapshot summary" of the clinical features, etiology, diagnostic methods, and preferred treatment for the disease to be discussed.

Dr Bartlett states that he intends the text to "provide a practical management approach to common clinical conditions." To that end, the book focuses on important clinical issues such as factors influencing the decision to hospitalize a patient with pneumonia, the marked benefit of early antibiotics for pneumonia, management of pleural effusions in the setting of pneumonia, and the rationale for treating (or not) pharyngitis with antibiotics. Despite the pragmatic emphasis, the book summarizes a large amount of clinical data to support recommendations or to illustrate the lack of evidence supporting currently accepted clinical practice. In addition, the author provides interesting historical perspectives at several points throughout the text. For example, the technique of transtracheal aspiration is discussed in some detail, as are a number of clinical studies using the technique. This combination of old and new data is a refreshing approach that makes the book extremely readable. Equally appealing, the concise writing and good summary tables will allow even busy clinicians to quickly read the book over a few sittings.

Although the book is intended for "practitioners," the audience for whom it will prove beneficial should be further clarified. The first chapter, "Pneumonia," represents over half of the total material discussed. It covers community-acquired pneumonia, nosocomial pneumonia, aspiration pneumonia, and pneumonia in the immunocompromised host. In addition, there is a short section addressing empyema. Physicians doing large amounts of inpatient work will be unlikely to discover new information regarding treatment here, though the historical perspective makes for interesting reading and recent clinical guidelines and studies are well summarized. On the other hand, physicians who rarely treat pneumonia; medical students doing medicine, pulmonary, or infectious disease clerkships; acute care and intensive care unit nurses; and respiratory therapists will probably find this chapter to be a practical, concise, and informative review of these common causes for inpatient admission. Physicians and mid-level practitioners who practice predominantly outpatient medicine will find the short section discussing site of treatment (inpatient vs outpatient) beneficial.

The final four chapters of the text cover a variety of conditions typically treated in the outpatient setting, including cough syndromes, the common cold, streptococcal pharyngitis, and sinusitis. Given the current widespread misuse of antibiotics in outpatient medicine, this portion of the book will provide either important new education or a reminder to any practitioner about the limited role of antibiotic therapy in these conditions. I recommend this as required reading for any primary care provider.

As mentioned above, this book is a pocket-sized paperback that is highly readable.

The font chosen for the main text is appropriately sized for reading without strain. In addition, the tables are well organized and provide important details such as antibiotic dosing, summaries of important clinical studies, differential diagnostic considerations, and microbiologic data. I found only two minor typographical errors, both within tables. The references are current and extensive, allowing easy access to the primary references on which recommendations are based. Finally, with a price of $14.95, the book has a favorable utility/cost ratio.

I have only a few criticisms to make of Dr Bartlett's book. First, though generally up to date, it is not surprising that there are a few places in which this new book is already outdated. The most obvious of these is encouragement of the use of sucralfate rather than H_2 blockers for stress ulcer prophylaxis on the basis of lower rates of nosocomial pneumonia. Though previously thought to be the case, more recent studies call that conclusion into question.

Second, in an effort to remain current, the author cites the potential utility of neuraminidase inhibitors in the treatment of influenza, based on preliminary clinical studies available at the time the book was written. However, considering the potential for misuse of these agents (ie, use for viral upper respiratory infections), their high cost, and what appears to be marginal clinical utility, deferring comment would have been more consistent with the author's hope of discouraging antibiotic use in situations of dubious clinical benefit.

Finally, technical details in the text occasionally seem to exceed its intended scope. For example, the techniques of transtracheal aspiration and several approaches to attaining quantitative bacterial cultures via bronchoscopy are discussed in quite specific detail. Though interesting, most readers of this text are unlikely to be performing these techniques. As a result, these discussions could be more general, allowing the excellent chapter on pneumonia to be even more concise.

In summary, this is a concise, easily read book that will prove beneficial to a wide audience of people involved in the care of patients with infections of the respiratory system. While it contains minimal original information, it represents an excellent and up-to-date summary of a tremendous amount of clinical data. Though it probably serves the primary care provider best, I suspect that nursing and medical students, nurses, and respiratory therapists will also find it valuable.

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Near Misses in Pulmonary and Cardiothoracic Critical Care. Joseph Varon MD, Garrett L Walsh MD MSc, Robert E Fromm Jr MD MPH. Philadelphia: WB Saunders. 1999. Softcover, illustrated, 120 pages, $27.50.

As organized medicine begins to focus more intently on medical errors and diagnostic/therapeutic misadventures, the analysis of "near misses" has gained in importance. In the most traditional sense, such events provide important diagnostic and therapeutic lessons for the medical community, serving as poignant reminders that help us avoid making the same mistake twice. In **Near Misses in Pulmonary and Cardiothoracic Critical Care**, the authors have produced a collection of 30 clinical vignettes intended to provide such lessons for the readership. The ideal readership would include practicing critical care physicians of all specialties, respiratory therapists, critical care nurses, and emergency room staff. Medical residents, critical care fellows, and respiratory therapy students might also be interested.

While the clinical cases are easily readable and at times entertaining, in the final analysis the collection falls short of its intended aim. In reviewing the 30 cases, I categorized only 6 cases (1, 2, 6, 14, 18, and 20) as true near misses. The other cases were not near misses at all, but either just interesting clinical vignettes, examples of clinical misadventures—common medical procedures gone awry—or complete misses where the providers literally missed the diagnosis totally. The accompanying discussions are well referenced but less than exhaustive.

By way of example, Case 12, "A Tall Man with Thick Glasses and Chest Pain," is that of a 30-year-old man evaluated by the medical system because of chest discomfort experienced while playing basketball. This tall, thin patient is initially considered to have acute myocardial infarction based on electrocardiography. The initial chest radiograph is not described. Lytic therapy is administered. Aortic dissection is considered only after pain worsens and the patient becomes hypotensive. Even after echocardiogram shows pericardial effusion with right ventricular diastolic collapse, the patient still requires cardiac catheterization to establish the diagnosis of proximal aortic dissection with cardiac tamponade before going to the operating room for repair of the dissection. Though the patient survived, this case must be considered a "complete miss" based on the absence of consideration of this important diagnostic entity at the outset and the administration of lytic therapy.

In another vignette, Case 20, "New Murmur and Collapse in an Investment Banker Admitted to the Coronary Care Unit," a 38-year-old male experiences increased shortness of breath in association with a new systolic murmur 24 hours after admission for an anterior myocardial infarction. This diagnosis is neither overlooked nor preventable. As described, the patient is treated appropriately with resultant repair of the ventricular septal defect. While this case is interesting and complications of acute myocardial infarction are important for all practicing intensive care physicians, the only lesson is simply the reminder of potential post myocardial infarction complications. Free-wall rupture and papillary muscle rupture are also mentioned, but there is no substantial description of the management or clinical presentation of these entities to serve as sound instruction for the reader.

In Case 28, a Swan-Ganz catheter fragment becomes lodged in the right heart after an inexperienced resident cuts the catheter at the skin in order to change it over a guidewire. This type of mistake should never happen in a critical care unit run by experienced clinicians. It represents a simple therapeutic misadventure resulting in a potentially complicated and costly effort by invasive radiology to retrieve the catheter fragment. The authors may have missed an opportunity to reinforce the most important clinical lesson from this case—emphasizing the use of cordis introducers for Swan-Ganz catheters and avoidance of cutting any catheter at the skin for fear of loss of control of the catheter and resultant embolization.

Despite falling short of its potential, the collection of clinical cases is relatively well written and is a quick read. In general, the material is entertaining and free of important errors. One rare error is noted on Page 105. A patient with a 4 cm spiculated mass in the left upper lobe is described as "Stage T2." This terminology is confusing because the stage needs to be based not only on tumor size (T_{0-4}) but also on nodal status

(N_{0-3}) and the presence or absence of distant metastasis (M_0 or M_1). This patient, as presented, is Stage I (T_2, N_0, M_0). On the positive side, each of the cases was interesting and each possessed a lesson regardless of whether they represented a misadventure, simply an interesting case, a near miss, or a complete diagnostic misadventure.

The book is presented in an attractive softbound cover and is accompanied by several interesting figures. As is common, some of the radiographs reproduced poorly and the findings were difficult to discern.

We must all learn from our mistakes. Indeed, near misses can be instructive for intensive care unit care providers. While a true collection of near misses would have been well received, the authors are to be congratulated for taking on a culture in Medicine where mistakes are often ignored for fear of reprisal. The authors remind us that a mature approach to dealing with mistakes—and learning from them—is the best way to avoid the same pitfalls in the future and to improve the quality of care.

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Medically Speaking: A Dictionary of Quotations on Dentistry, Medicine and Nursing. Selected and arranged by Carl C Gaither and Alma E Cavazos-Gaither. Bristol, United Kingdom, and Philadelphia: Institute of Physics Publishing. 1999. Softcover, illustrated, 481 pages, $39.

John Bartlett published the first edition of *Familiar Quotations* in 1855, which became the archetype for all subsequent collections. Sixteen editions of Bartlett have followed; there are now collections of political, historical, religious, literary, and humorous quotations, along with ones from television and the movies. Naturally there are books with specific quotations about lawyers and doctors. All of these collections bespeak of a greater reader interest in the genre than I would have expected. The basis of this interest is not at all clear, as it seems to me that such books are chiefly for occasional browsing; others have, however, pointed out that they can be a worthwhile reading experience. A good quotation book can be a compendium of intellectual and cultural history as well as a source of insight into changing mores. Such a view may suit larger tomes that are more broadly inclusive, but less so for those collections more narrowly focused.

Regardless of the subject addressed, quotations should cover the past as well as the present, so as to enrich our understanding of changes in outlook, opinion, standards, and philosophy—not just changes that have occurred, but those ideas and ideals that have remained unchanged, pointing to the verities on which they are based. Really memorable quotations should have profundity balanced with humor—wisdom with wit. Though some collections pride themselves on the familiar, critics warn of over-repeating the familiar. The familiar over time becomes the hackneyed. Avoiding the bar set by familiarity is but one obstacle for the collector of quotations. Quotations are effective rhetorical devices but can be a shabby substitute for thinking. Emerson wrote (and I *quote!*), "I hate quotation, tell me what you know." On the other hand, with circumspection, a more positive use can be made of a quotation. Martin Boorstin pointed out: "With a quotation we make it plain that we are letting someone else who says it better, say it for us."

An arranger's task of selection is made difficult by the absence of any clear definition of a quotation. Published collections probably include maxims, analects, epigrams, quips, poems, limericks, jokes, or paronomasia. Selection perforce becomes subjective. My own preference is for good, memorable quotes to have concision, be pithy, and contain some wit or whimsy. I eschew banality, entries so prolix that the point is lost, or those lacking any irony. The identity of an author of a quotation of renown is of interest, but I have found that many "anonymous" authors or persons I have never heard of have contributed memorable quotations. It seems that a great deal of effort is expended by some arrangers and editors to check the authenticity of (especially older) quotations, but to me this is often unnecessary; I am reminded of Dr Johnson's observation that composers (of epitaphs) were never under oath. In the preface of his book *Familiar Medical Quotations*, Maurice Strauss quoted a standard (for books of quotations) written by Lewis Galantière.[1]

> A truly serviceable book of quotations is not an expression of its compiler's preferences. . . . It has no preconceptions, takes no stand, is fair to all doctrines and all peoples. The true, the false, the debatable; the tragic and the comic; the wise and the witless come within its compass. A book of quotations is an orderly assembling of those lines. . . which mankind has found itself unable to forget—better yet, to do without. It is a *summum* of human experience.

This is a fine ideal, but I cannot accept that one working within so broad a compass can be objective.

The authors of **Medically Speaking** have assembled a collection of quotations related to the professions of medicine, dentistry, nursing, and pharmacy, addressed to students and practitioners of those professions as well as lay persons interested in health care. The appearance of the book is striking but attractive, and, as the editors state, of a size that can unobtrusively sit on an office desk. There is a cartoon on the cover that suggests a lighthearted tone to the book. Personally, I would not put this book on my desk while interviewing patients who might mistakenly infer a prevailing lack of *gravitas.* The print is of a size quite suitable even for my presbyopia and the entries are well set apart for even greater ease of reading. On this point the publisher scores, as I have experienced difficulty trying to read the tightly packed text in other books. Each topic begins on a separate page and those instances where there is only one short entry for a topic, the resulting half-empty page seems a waste. A subject and author index as well as a complete bibliography complete the text.

This book must be compared with two other collections of medical quotations; both contain entries related to nursing, dentistry and pharmacy. *Familiar Medical Quotations*[2] edited by Maurice Strauss is a large book, comparable in size to *Bartlett's*, and suffering from being over 30 years old—lacking references to the many innovations and developments that have taken place since that time. The second, *Medical Quotes–A Thematic Dictionary*,[3] edited by John Daintith and Amanda Isaacs is a book closer in size to **Medically Speaking** and closer in date of publication. Because this work is similar in size and scope, it is the one that I shall compare with **Medically Speaking**, when it seems appropriate.

Medically Speaking has over 1,500 entries, and this relatively small number requires that the editors be selective and, if possible, avoid entries so familiar they qualify as hackneyed; also selections should cover times in the distant as well as recent past. I believe they have largely achieved that. To set theirs apart from other collections, it behooves them to pick quotations highlighting the times since earlier publications. Both Strauss and Daintith published their works either before the AIDS epidemic or early on in its development. Daintith offers 4 entries on the subject, while **Medically Speaking** has none. Today alcohol is a major health and social problem and while Daintith gives 43 quotes on alcohol and drinking, **Medically Speaking** has none. Daintith and Isaacs have sections on "Eating," "Healthy Eating," and "Obesity," while **Medically Speaking** only has a section on "Nutrition" with but 4 entries. "Alternative (or Integrative) Medicine" and "Holistic Medicine" are popular as well as controversial topics today and Daintith and Isaacs give 13 quotes on "Holistic Medicine," versus none in **Medically Speaking**. Other topics that reasonably should be included but aren't, though they are in Daintith and Isaacs, include "Euthanasia" (7), "Medical Jargon" (21), and "Medical Ethics" (5). Also missing are entries for "Neurosis," "Depression," "Anxiety," "Psychosis," or "Psychiatry." Instead of finding any one of these headings, I was disappointed to note one page "dedicated" to the subject of "Nausea" with but one banal quotation; this facing another full page on the subject of "Nerve" with but one quote of similar quality.

Another failing of this book is the absence of quotes relating to other health care professionals. In the preface, Gaither and Cavazos-Gaither state a desire to address their collection to students of the medical, dental, nursing, and pharmacy professions, overlooking the large number of other professionals in health care. Today we celebrate the skills of radiological, respiratory, and operating room technicians as well as nutritionists, physical therapists, and social workers. It is unlikely that each of these groups would be a source of interesting quotations, but surely there are some; if not, some recognition of their role in health care should be included—at least in the preface.

Medically Speaking does have interesting sections and original (at least to me) quotations. The section "Nouns of Multitude" consists of a lengthy list of humorous names for medical specialists. I have not seen such a listing in any other collection, and though one might question whether it meets the criteria of a quotation (whatever that may be), I found it mildly humorous. Another unique section was "Medical Mnemonics." I recall that mnemonics did play a role as a learning tool for some medical students, especially when studying anatomy. One of my fellow students had a small leather-bound book filled with mnemonics for just about every part of the human body. I never saw such a book again, which is just as well since many of the "quotations" were unfit to print, even in these sexually uninhibited times. I can still recall only two and recognized at the time, as did most students, that it was usually easier to memorize the anatomy than the mnemonic. One I remember was: "*O*n *O*ld *O*lympus' *T*owering *T*op, *A F*renchman *A*nd *G*erman *V*iewed *A H*op." With this encausticated in your cerebral cortex, you could remember the twelve cranial nerves. (You had, of course, to remember the names and correct sequence of the nerves the initial 3 "O's" stood for!) The second one I remember is presented in the book and is the only entry with which I can quibble (and it is a quibble). To recall the names of the bones of the hand, one has only to remember: "*N*ever *L*ower *T*illie's *P*ants. *G*rand*M*other *M*ight *C*ome *H*ome." "*G*rand*M*other" serves to recall the greater multangular bone. I learned it as "*G*rand*M*other *L*ucy *M*ight *C*ome *H*ome," thus recalling the lesser multangular. This was one of two very small corrections I would make in the book; the other is a quote referenced to John Bryce (p 204) who was probably Sir James Bryce (1838–1922).

There are many good quotations throughout the book, and one section, "Teaching," though having only 11 entries, offers a good mix of the humorous and the cynical and all give the reader something to think about. The section on "Physicians" contains, not surprisingly, a large number of quotes attributed to Sir William Osler as collected by Harvey Cushing, which will probably be familiar to medical students and practitioners; they are of particular interest to me for their depiction of the ideal physician. They limn an individual who is omniscient, lives a life of faithful dedication and self-sacrifice that may be seen as both Spartan and monastic, hardly the portrait of either a contemporary physician or, sadly, an ideal felt by many necessary to aspire to. Were there really physicians like that?

Many well-known authors are represented in this collection, and those such as Mark Twain, GK Chesterton, and Ambrose Bierce live up to their reputations as authors of marvelous quotations. I was disappointed in the selections from William Bean's "Aphorisms from Latham." Peter Mere Latham (1789–1875) was a poet and essayist and William Bean was Professor of Medicine at the University of Iowa, a great teacher and writer, but I found his many selections from Latham unusually bland. I was moved, however, by a quotation by Flannery O'Connor (1925–1964):

> I have never been anywhere but sick. In a sense sickness is a place, more instructive than a long trip to Europe, and it's always a place where there's no company, where nobody can follow. Sickness before death is a very appropriate thing and I think those who don't have it miss one of God's mercies.

What is lacking in wit here is made up for with profound understanding. This by a young woman who was deeply religious and whose spiritual strength sustained her through years of suffering from lupus.

Medically Speaking is an enjoyable book; however, I do not feel it measures up to the one by Daintith and Isaacs.[3] The latter book is roughly the same size, but has far more entries and covers more of the health care profession. Though published 11 years ago it contains quotations that are more current than those in **Medically Speaking**. I did enjoy reading **Medically Speaking** and believe, to paraphrase TS Eliot, it can be read by many for pleasure and profit.

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Just Quit—Online!

There are many smoking cessation programs sponsored by health care providers that provide face-to-face support. There are also a number of online locations that attempt to provide the same service on the Internet. These various resources provide tips, motivation, and steps to quit. Some also provide a community of support from fellow quitters. Online support has the advantage of being available any time, so that a person with a craving at 4 a.m. may find a fellow quitter to turn to simply by logging in. A few are highlighted here.

QuitNet.com was established as a free service provided by the Boston University of Public Health. The site provides "proven scientific methods . . . to deliver support to smokers whenever they need it." There are quitting tips and tools, including a quiz to determine your reasons for smoking and to see how prepared you are to quit.[1]

During the review period (September 1–5, 2000), there were usually 30 to 60 members signed in. The site requires free registration in order to make use of all features. The privacy policy does not allow any information to be shared without explicit permission. It also goes one step further and will cancel an account and delete your information upon request. The personal profile can reveal as much, or little, information as desired.

Once you select your quitting date, you may elect to receive weekly e-mail guidelines up to the final 14 days of smoking. From that date on, you get daily tips until your quit date and for two weeks after. The site offers a database of local programs in case there is additional need for face-to-face support. In the Kansas City metro area, for example, there were 20 different providers listed.

Registered members are offered 5 different options: "Talk," "Messages," "People," "Tools," and "Gadgets." "Talk" provides discussion groups that cover the quitting process, milestones, weight management, and depression. In addition, it offers access to smoking cessation counselors who provide expert advice on quitting problems. "Messages" is an area for private e-mail exchanges. "People" shows a list of online users and allows you to contact them. "Tools" helps determine where you are in the quitting process. The most entertaining item is "Gadgets," which provides an up-to-the-minute count of how many cigarettes you haven't smoked, how much money you have saved, and how much of your life you have saved.

The members seem to be constantly involved in supporting each other. A review of past dialogues showed no criticism of failed quitters; instead, they encouraged the fellow quitter to keep trying without making any judgements. This site would be ideal for motivated people who have already decided to quit.

The Australian National Tobacco Campaign (ANTC) takes a harder approach to quitting. The home page greets you with a graphic image of a blocked airway.[2] The reasoning behind this, and all of their other images, is to produce a strong, emotional response and to let smokers know that every cigarette is doing damage, even though they may not feel anything. They stress that smoking is not a future risk, it has immediate implications. The site is part of a broad national campaign that started in 1997 and includes print, radio, and television ads.

An online assessment is available for smokers to determine readiness to quit. It was too long for casual inquiries, compared to the shorter Fagerstrom quiz. The multiple parts took about 15 minutes to complete.

The ANTC does not offer online support, but does include numerous contacts and a quitting guidebook for Australian residents. Information for quitting follows traditional methods. This site may be an appropriate referral for a smoker willing to gamble with their future health.

Other sites ranged from personal diaries of the quitting experience,[3] to programs with a cartoon quality aimed at helping teens to quit.[4,5] The most amusing graphic was a convulsing mailbox that asked fellow COPDers to "cough up a letter."[6]

An entertaining result of a search for quitting sites was a set of "three unfiltered tales" by Stephen King.[7] In these stories, he very accurately describes the phases of withdrawal: "the first two weeks, the physical period, with headaches, twitches, nausea and irritability; and the longer, mental period of depression mourning, forgetfulness, dyslexia . . . and a feeling of mild unreality." The difficulties of nicotine withdrawal are superimposed on King's artful stories of an unexpected divorce, a stay in a haunted hotel room, and a tortured captive who uses his last cigarette in an attempt to save his life.

King, an ex-smoker, knows first-hand of the pains of withdrawal. He was forced to quit following an accident in which he was hit by a van while walking along a road and spent a lengthy recovery period in the hospital. He refers to it as the "Dodge van cure," and, while it was very effective, he would not recommend it as a preferred method.

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Medscape
(reviewed June 13—July 7, 2000)

Medscape (*www.medscape.com*) was established with the intent of being an authoritative online resource for medical professionals. Compared to many other sites, they have succeeded. The site is clearly aimed at medical professionals, covering aspects from patient care and treatment to medical business management. **Medscape** is part of MedicalLogic. The parent company also owns and manages CBSHealthWatch, a general health information resource intended for the public, and *www.98point6.com*, a site for consumers to maintain their own health records.

Medscape is a free service that requires registration to take advantage of its various functions. When signing up for membership, you may designate a preference for your **Medscape** home, the respiratory care section being one of about 20 options. Each of the interest areas has its own starting point. The home page for each area provides a few daily headlines of interest, along with access to related areas on the Web site. As expected with a free service, advertising is ubiquitous, but does not significantly slow down page loading times. The privacy policy does not allow membership information to be distributed to third parties without express consent.

One advantage to reviewing traditional materials is that the entire item can be observed: one can easily determine the page length of a book or the minute length of a videotape. Web-based resources, however, do not provide any indicator of size. During the review process, whole areas within **Medscape** were discovered serendipitously, but an attempt to return to a particular page could result in lengthy trips with dead-ends and backtracks. The site index was limited to the specialty area being viewed. Fortunately, the content was interesting enough to keep you wandering around. Unfortunately, many hours were wasted in the wandering during the review.

The **Medscape** respiratory care section home page starts with main sections consisting of headlines, conference summaries, treatment updates, journal room, and exam room. Headlines are extracted daily from Reuters News Service. Most of the news items during the review period were relevant, useful, or at least interesting. There were occasional nuggets, such as an abstract on June 19, 2000, showing that plastic soda bottles were as effective for aerosol delivery as AeroChamber and BabyHaler, and less expensive.[1] Other items covered such topics as new drug availability, treatment protocols, and medical industry acquisitions.

Treatment updates provide reviews of therapeutic regimens. They are written by a host of **Medscape** authors. An archive also provides access to many of the American Association for Respiratory Care Clinical Practice Guidelines, which are conveniently sorted into logical groups such as aerosol delivery, asthma, and critical care. In addition to the treatment updates, **Medscape** provides access to the Cochrane Review Abstracts, a widely respected source that provides summaries of treatment data gathered from clinical trials.

Conference highlights ranged from the Second International Symposium on Influenza and Other Respiratory Viruses (December 1999) to the 96th International Conference of the American Thoracic Society (May 2000). Each of the conference highlights included "Expert-Authored Reports" that summarized sessions. The summaries seemed to include keynote or invited speakers and plenary sessions of general interest. Daily summaries were posted during the meetings. Of the four conferences listed during the review, three offered continuing education credits for reviewing the proceedings.

The journal room offers articles written specifically for the various **Medscape** specialty areas. Most articles feature small versions of graphs or pictures that can be clicked on to provide more useful-sized images (this helps to minimize page loading time). References for most articles are conveniently displayed in a separate window, obviating the need to flip back and forth through Web pages. The articles are also available in a printer-friendly format to allow them to be printed and read at leisure. During the review period this section featured a discussion on the therapeutic role of nitric oxide in respiratory disease.[2] The article fell far short of the comprehensive quality of recent review articles in RESPIRATORY CARE.[3] For example, it included a graph consisting of two lines with no explanation.[4] A pink line shows a bimodal distribution of "Ventilation or Perfusion (L/min)" versus Ventilation/Perfusion Ratio, while a blue line shows one peak greatly reduced. No indication is made of whether either line shows a disease state. A caption was later added that still did not clarify the data. One feature missing from the journal room is a discussion area to raise issues with the authors, such as the above-mentioned graph.

The source of the article, *Medscape Respiratory Care: a Medscape eMed Journal*, describes itself as a peer-reviewed electronic journal available only in an online version. A list of the journal issues reveals that it is usually published every two months. The content of each issue varies from a single case presentation to multiple articles. Recent articles have covered a wide gamut of respiratory topics, such as aspergillosis, pneumonia, pregnancy, ventilator-associated pneumonia, and latex sensitivity. In addition, there are numerous short case studies that include radiology, histology, or ultrasound data. Many of the longer articles provide Category 1 continuing education credit. There are additional **Medscape** journals covering other specialties.

The journal room also includes Micromedex CareNotes. CareNotes are edited and provided by MicroMedex, not **Medscape**. These are intended to provide patient education in an easy-to-understand format. While they are written in simplistic terms, the information is at best misleading or incomplete. For example, chronic obstructive pulmonary disease and chronic bronchitis are lumped together as one disease entity. This causes confusion when signs of chronic obstructive pulmonary disease are described as "cough for 3 months of a year for 2 years in a row." CareNotes should not be recommended to patients until appropriate staff have reviewed them.

Continuing education programs offered on **Medscape** are approved by the Accreditation Council for Continuing Medical Education. Various organizations sponsor most of the articles, providing continuing education units free of charge. There are a few exceptions for which a small fee is required. A certificate can be printed upon successful completion of a test by a registered user to meet continuing education requirements. Not all states accept these credits for respiratory practitioners. For example, a check with the Kansas Board of Healing Arts did not provide a definitive answer. There was some skepticism because of the newness of the medium. One staff person suggested that specific requests would have to be received before they would make a determination.

The exam room presents clinical findings and brief discussions of diagnosis and treatment. Many of the offerings in the exam room are similar in nature to those found in **Medscape**'s respiratory care section articles. During the review period no new exams were added to the three that were listed.

The library and journal areas also seem to be used interchangeably. Between the two, they provide access to excerpts of popular journals, newsletters, and online textbooks, all but two of which are published by Professional Communications Inc. The journal excerpts did not seem to follow any predictable pattern. Merging the library and journal sections would also eliminate the need to search in both places for information.

Medscape offers access to the MEDLINE database, and articles found in the search can be ordered from KnowledgeFinder, for a rather stiff fee. Most articles cost from $20–35 for delivery in several weeks. Costs climbed quickly for more expeditious service. A reasonable alternative is also available for a small number of journals. Dow Jones Publications library includes a number of relevant, full-text medical publications. The site claims over 500 publications in the "medical and health" category: useful journals are more in the range of 50. The articles are only $2.95 each and are delivered online.

Article references of interest from MEDLINE searches may be marked for downloading. **Medscape** provides formatting in many of the popular citation formats, including Medlars, Bookends, Endnote, Papyrus, Procite, Refman, and QuickDoc. The results can also be sent by e-mail using either QuickDoc or Medlars format.

Medscape includes an online bookstore with over 90,000 titles. Unlike most other online bookstore links, this one is not connected to amazon.com. Few of the common respiratory care titles listed at amazon.com are discounted. The **Medscape** bookstore not only provides significant discounts (Burton's *Respiratory Care* was 10% off), but they had several discounts offers worth up to $100 off an order.

Several smaller sections held interesting material. A job center is included with positions listed in all practice fields. There were usually 30–40 respiratory therapist jobs listed. A facility is also provided for job seekers to post their curriculum vitaes. Another section provides medical humor with a daily cartoon and humorous writings. The last section offers personal financial management.

While there are still a few weak areas, the site is very useful to respiratory therapy students and educators with its many case presentations. It adds value by offering continuing education credit at no cost to respiratory therapists. Lastly, access to MEDLINE and other similar databases should make **Medscape** a frequently used resource in many respiratory care departments.

Steven B Nelson MS RRT
Overland Park, Kansas
Mayo Pulmonary Services
Rochester, Minnesota

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4. http://respiratorycare.Medscape.com/Medscape/RespiratoryCare/journal/2000/v04.n02/mrc4924.hige/mrc4924.hige-07.html

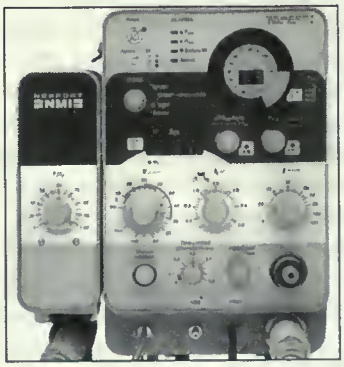
New Products & Services

News releases about new products and services will be considered for publication in this section. There is no charge for these listings. Send descriptive release and glossy black and white photographs to RESPIRATORY CARE, New Products & Services Dept, 11030 Ables Lane, Dallas TX 75229-4593. The Reader Service Card can be found at the back of the Journal.

Home Disinfection Kit. Maril Products Inc has created a Home Care Kit containing a two-quart bucket, test strips, a measuring cup, and a 2-oz bottle of Control III® disinfectant. Maril says their product is more effective than vinegar and is 8 times more cost-effective. And now the company is offering an introductory training package for Control III surface and instrument home disinfecting products. The package includes educational patient materials describing product applications and usage; product samples with patient instructions are also included. The company offers this training kit at no charge. For more information from Maril Products Inc, circle number 174 on the reader service card in this issue, or send your request electronically via "Advertisers Online" at http://www.aarc.org/buyers_guide/

Blood Pressure Monitor. Medwave Inc. has received FDA 510(k) approval for its handheld blood pressure monitor, Vasotrax™. According to Medwave, the Vasotrax is a 3.5 oz. wireless, handheld device that measures systolic and diastolic blood pressures as well as pulse rate from the radial artery on the wrist. The company reports that clinical studies showed the device had a mean correlation 0.9 compared to an indwelling arterial catheter. For more information from Medwave Inc, circle number 175 on the reader service card in this issue, or send your request electronically via "Advertisers Online" at http://www.aarc.org/buyers_guide/

Continuous Nebulizer Kits. B&B Medical Technologies introduces its new continuous nebulizer kit configurations. The Adult HOPE kit contains a HOPE nebulizer, 6 feet of large bore tubing and an adult aerosol mask (#11320). The Pediatric HOPE kit contains a HOPE nebulizer, 6 feet of large bore tubing and a pediatric aerosol mask (#11330). According to the company, these two kits contain everything the practitioner needs to rescue the severe pulmonary patient without having to waste valuable time locating accessories. For more information from B&B Medical Technologies, circle number 176 on the reader service card in this issue, or send your request electronically via "Advertisers Online" at http://www.aarc.org/buyers_guide/



Ventilator. Newport Medical Instruments Inc has released the Newport E100M. The company says the device is capable of ventilating neonates, pediatrics, and adults, and is suitable for emergency room, intrahospital transport, and post-acute/long-term settings. Newport describes the new ventilator as compact and versatile with an internal battery (up to 8 hours operation) and says it features auto-set alarms and back-up ventilation for patient safety.For more information from Newport Medical Instruments Inc, circle number 177 on the reader service card in this issue, or send your request electronically via "Advertisers Online" at http://www.aarc.org/buyers_guide/

Allergy Relief Medication. AstraZeneca has recently made available nationwide a new nasal allergy relief medication called Rhinocort Aqua™ (budesonide). The company says this nasal spray is designed for the treatment of seasonal allergies and perennial allergic rhinitis in adults and children six years of age and older. According to AstraZeneca, Rhinocort is the first nasal steroid medication for children and adults with a starting dose of only one spray per nostril, once each day. For more information from AstraZeneca, circle number 178 on the reader service card in this issue, or send your request electronically via "Advertisers Online" at http://www.aarc.org/buyers_guide/

www.rcjournal.com

call for abstracts

RESPIRATORY CARE • OPEN FORUM 2001

The American Association for Respiratory Care and its science journal, RESPIRATORY CARE, invite submission of brief abstracts related to any aspect of cardiorespiratory care. The abstracts will be reviewed, and selected authors will be invited to present posters at the OPEN FORUM during the AARC International Respiratory Congress in San Antonio, Texas, December 1-4, 2001. Accepted abstracts will be published in the October 2001 issue of RESPIRATORY CARE. Membership in the AARC is not required for participation. All accepted abstracts are automatically considered for ARCF research grants.

SPECIFICATIONS—READ CAREFULLY!

An abstract may report **(1) an original study, (2) the evaluation of a method, device or protocol, or (3) a case or case series.** Topics may be aspects of adult acute care, continuing care/rehabilitation, perinatology/pediatrics, cardiopulmonary technology, or health care delivery. The abstract may have been presented previously at a local or regional—but not national—meeting and should not have been published previously in a national journal. The abstract will be the only evidence by which the reviewers can decide whether the author should be invited to present a poster at the OPEN FORUM. Therefore, *the abstract must provide all important data, findings, and conclusions.* Give specific information. Do not write such general statements as "Results will be presented" or "Significance will be discussed."

ESSENTIAL CONTENT ELEMENTS

Original study. Abstract *must* include (1) Background: statement of research problem, question, or hypothesis; (2) Method: description of research design and conduct in sufficient detail to permit judgment of validity; (3) Results: statement of research findings with quantitative data and statistical analysis; (4) Conclusions: interpretation of the meaning of the results.

Method, device, or protocal valuation. Abstract *must* include (1) Background: identification of the method, device, or protocol and its intended function; (2) Method: description of the evaluation in sufficient detail to permit judgment of its objectivity and validity; (3) Results: findings of the evaluation; (4) Experience: summary of the author's practical experience or a lack of experience; (5) Conclusions: interpretation of the evaluation and experience. Cost comparisons should be included where possible and appropriate.

Case report. Abstract *must* report a case that is uncommon or of exceptional educational value and must include (1) Introduction: relevant basic information important to understanding the case. (2) Case Summary: patient data and response, details of interventions. (3) Discussion: content should reflect results of literature review. The author(s) should have been actively involved in the case and a case-managing physician must be a co-author or must approve the report.

FORMAT AND TYPING INSTRUCTIONS

Accepted abstracts will be photographed and reduced by 40%; therefore, the size of the original text should be *at least* 10 points. A font like Helvetica or Times makes the clearest reproduction. The first line of the abstract should be the title in all capital letters. Title should explain content. Follow title with names of all authors (including credentials), institution(s), and location; underline presenter's name. Type or electronically print the abstract *single spaced in one paragraph on a clean sheet of paper, using margins set so that the abstract will fit into a box no bigger than 18.8 cm (7.4") by 13.9 cm (5.5"), as shown on the reverse of this page.* Insert only one letter space between sentences. Text submission on diskette is allowed but must be accompanied by a hard copy. *Data may be submitted in table form, and simple figures may be included provided they fit within the space allotted. No figure, illustration, or table is to be attached to the abstract form.* Provide all author information requested. Standard abbreviations may be employed without explanation; new or infrequently used abbreviations should be spelled out on first use. Any recurring phrase or expression may be abbreviated, if it is first explained. Check the abstract for (1) errors in spelling, grammar, facts, and figures; (2) clarity of language; and (3) conformance to these specifications. An abstract not prepared as requested may not be reviewed. Questions about abstract preparation may be telephoned to Linda Barcus at (972) 406-4667.

Early Deadline Allowing Revision. Authors may choose to submit abstracts early. Abstracts postmarked by May 31, 2001 will be reviewed and the authors notified *by letter only* to be mailed by June 15, 2001. Rejected abstracts will be accompanied by a written critique that should, in many cases, enable authors to revise their abstracts and resubmit them by the Final Deadline (July 17, 2001).

Final Deadline. The mandatory Final Deadline is July 17, 2001 (postmark). Authors will be notified of acceptance or rejection *by letter only.* These letters will be mailed by September 1, 2001.

Mailing Instructions. Mail (Do not fax!) 2 clear copies of the completed abstract form, diskette (if possible), and a stamped, self-addressed postcard (for notice of receipt) to:

2001 RESPIRATORY CARE OPEN FORUM
11030 Ables Lane
Dallas TX 75229-4593

submit your OPEN FORUM abstract electronically
visit www.rcjournal.com

RESPIRATORY CARE OPEN FORUM 2001 Abstract Form

18.8 cm or 7.4"

13.9 cm or 5.5"

1. Title must be in all upper case (capital) letters, authors' full names and text in upper and lower case.
2. Follow title with all authors' names including credentials (underline presenter's name), institution, and location.
3. Do not justify (ie, leave a 'ragged' right margin).
4. **Do not use type size less than 10 points.**
5. All text and the table, or figure, must fit into the rectangle shown. (Use only 1 clear, concise table or figure.)
6. Submit 2 clean copies.

Mail original & 1 photocopy (along with postage-paid postcard) to

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American Association for Respiratory Care

MEMBERSHIP APPLICATION

Please read the eligibility requirements for each of the classifications in the right-hand column, then complete the applicable section. All information requested below must be provided, except where indicated as optional. See other side for more information and fee schedule. Please sign and date application on reverse side and type or print clearly. Processing of application takes approximately 15 days.

- ☐ Active
- Associate
  - ☐ Foreign
  - ☐ Physician
  - ☐ Industrial
- ☐ Special
- ☐ Student

Last Name ______________________________

First Name ______________________________

Social Security No. ______________________________

Home Address ______________________________

City ______________________________

State ____________ Zip __ __ __ __ __-__ __ __ __

Phone No. (______) ______________________________

Primary Job Responsibility (check one only)

- ☐ Technical Director
- ☐ Assistant Technical Director
- ☐ Pulmonary Function Specialist
- ☐ Instructor/Educator
- ☐ Supervisor
- ☐ Staff Therapist
- ☐ Staff Technician
- ☐ Rehabilitation/Home Care
- ☐ Medical Director
- ☐ Sales
- ☐ Student
- ☐ Other, specify ______________________________

Type of Business

- ☐ Hospital
- ☐ Skilled Nursing Facility
- ☐ DME/HME
- ☐ Home Health Agency
- ☐ Educational Institution
- ☐ Manufacturer or supplier
- ☐ Other, specify ______________________________

Date of Birth (optional) ______________ Sex (optional) ____________

U.S. Citizen? ____ Yes ____ No

Have you ever been a member of the AARC? ______________________

If so, when? From ________________ to ________________

AARC

Preferred mailing address: ☐ Home ☐ Business

For office use only

FOR ACTIVE MEMBER

An individual is eligible if he/she lives in the U.S. or its territories or was an Active Member prior to moving outside its borders or territories, and meets ONE of the following criteria: (1) is legally credentialed as a respiratory care professional if employed in a state that mandates such, OR (2) is a graduate of an accredited educational program in respiratory care, OR (3) holds a credential issued by the NBRC. An individual who is an AARC Active Member in good standing on December 8, 1994, will continue as such provided his/her membership remains in good standing.

PLEASE USE THE ADDRESS OF THE LOCATION WHERE YOU PERFORM YOUR JOB, NOT THE CORPORATE HEADQUARTERS IF IT IS LOCATED ELSEWHERE.

Place of Employment ______________________________

Address ______________________________

City ______________________________

State ____________ Zip __ __ __ __ __-__ __ __ __

Phone No. (______) ______________________________

Medical Director/Medical Sponsor ______________________________

FOR ASSOCIATE OR SPECIAL MEMBER

Individuals who hold a position related to respiratory care but do not meet the requirements of Active Member shall be Associate Members. They have all the rights and benefits of the Association except to hold office, vote, or serve as chair of a standing committee. The following subclasses of Associate Membership are available: Foreign, Physician, and Industrial (individuals whose primary occupation is directly or indirectly devoted to the manufacture, sale, or distribution of respiratory care equipment or supplies). Special Members are those not working in a respiratory care-related field.

PLEASE USE THE ADDRESS OF THE LOCATION WHERE YOU PERFORM YOUR JOB, NOT THE CORPORATE HEADQUARTERS IF IT IS LOCATED ELSEWHERE.

Place of Employment ______________________________

Address ______________________________

City ______________________________

State ____________ Zip __ __ __ __ __-__ __ __ __

Phone No. (______) ______________________________

FOR STUDENT MEMBER

Individuals will be classified as Student Members if they meet all the requirements for Associate Membership and are enrolled in an educational program in respiratory care accredited by, or in the process of seeking accreditation from, an AARC-recognized agency.

SPECIAL NOTICE — Student Members do not receive Continuing Respiratory Care Education (CRCE) transcripts. Upon completion of your respiratory care education, continuing education credits may be pursued upon your reclassification to Active or Associate Member.

School/RC Program ______________________________

Address ______________________________

City ______________________________

State ____________ Zip __ __ __ __ __-__ __ __ __

Phone No. (______) ______________________________

Length of program

- ☐ 1 year
- ☐ 2 years
- ☐ 4 years
- ☐ Other, specify ______________

Expected Date of Graduation *(REQUIRED INFORMATION)*

Month ________ Year ________

American Association for Respiratory Care • 11030 Ables Lane • Dallas, TX 75229-4593 • (972) 243-2272 • Fax (972) 484-2720

American Association for Respiratory Care MEMBERSHIP APPLICATION

Demographic Questions

We request that you answer these questions in order to help us design services and programs to meet your needs.

Check the Highest Degree Earned

- ☐ High School
- ☐ RC Graduate Technician
- ☐ Associate Degree
- ☐ Bachelor's Degree
- ☐ Master's Degree
- ☐ Doctorate Degree

Number of Years in Respiratory Care

- ☐ 0-2 years
- ☐ 3-5 years
- ☐ 6-10 years
- ☐ 11-15 Years
- ☐ 16 years or more

Job Status

- ☐ Full Time
- ☐ Part Time

Credentials

- ☐ RRT
- ☐ CRT
- ☐ Physician
- ☐ CRNA
- ☐ RN
- ☐ LVN/LPN
- ☐ CPFT
- ☐ RPFT
- ☐ Perinatal/Pediatric

Salary

- ☐ Less than $10,000
- ☐ $10,001-$20,000
- ☐ $20,001-$30,000
- ☐ $30,001-$40,000
- ☐ $40,000 or more

PLEASE SIGN

I hereby apply for membership in the American Association for Respiratory Care and have enclosed my dues. If approved for membership in the AARC, I will abide by its bylaws and professional code of ethics. I authorize investigation of all statements contained herein and understand that misrepresentations or omissions of facts called for is cause for rejection or expulsion.

A yearly subscription to RESPIRATORY CARE journal and *AARC Times* magazine includes an allocation of $11.50 from my dues for each of these publications.

NOTE: Contributions or gifts to the AARC are not tax deductible as charitable contributions for income tax purposes. However, they may be tax deductible as ordinary and necessary business expenses subject to restrictions imposed as a result of association lobbying activities. The AARC estimates that the nondeductible portion of your dues — the portion which is allocable to lobbying — is 26%.

Signature ______________________________

Date ______________________________

Membership Fees

Payment must accompany your application to the AARC. Fees are for 12 months. (NOTE: Renewal fees are $75.00 Active, Associate-Industrial or Associate-Physician, or Special status; $90.00 for Associate-Foreign status; and $45.00 for Student status).

☐ Active	$ 87.50
☐ Associate (Industrial or Physician)	$ 87.50
☐ Associate (Foreign)	$102.50
☐ Special	$ 87.50
☐ Student	$ 45.00
TOTAL	$________

Specialty Sections

Established to recognize the specialty areas of respiratory care, these sections publish a bi-monthly newsletter that focuses on issues of specific concern to that specialty. The sections also design the specialty programming at the national AARC meetings.

☐ Adult Acute Care Section	$15.00
☐ Education Section	$20.00
☐ Perinatal-Pediatric Section	$15.00
☐ Diagnostics Section	$15.00
☐ Continuing Care-Rehabilitation Section	$15.00
☐ Management Section	$20.00
☐ Transport Section	$15.00
☐ Home Care Section	$15.00
☐ Subacute Care Section	$15.00
TOTAL	$________

GRAND TOTAL = Membership Fee plus optional sections $________

- -

☐ Total Amount Enclosed/Charged $______________

☐ Please charge my dues (see below)

To charge your dues, complete the following:

- ☐ MasterCard
- ☐ Visa

Card Number

__ __ __ __ __ __ __ __ __ __ __ __ __ __ __ __

Card Expires ______/______

Signature ______________________________

Mail application and appropriate fees to:

American Association for Respiratory Care • 11030 Ables Lane • Dallas, TX 75229-4593 • (972) 243-2272 • Fax (972) 484-2720

MEDWATCH

THE FDA MEDICAL PRODUCTS REPORTING PROGRAM

For VOLUNTARY reporting
by health professionals of adverse
events and product problems

Page ____ of ____

Form Approved: OMB No. 0910-0291 Expires: 4/30/96
See OMB statement on reverse

FDA Use Only (Resp Cers)

Triage unit
sequence #

A. Patient information

1. **Patient identifier**
In confidence

2. **Age at time of event:** ____________
or
Date of birth:

3. **Sex**
☐ female
☐ male

4. **Weight**
____ lbs
or
____ kgs

B. Adverse event or product problem

1. ☐ **Adverse event** and/or ☐ **Product problem** (e.g., defects/malfunctions)

2. **Outcomes attributed to adverse event**
(check all that apply)
☐ death ____________ (mo/day/yr)
☐ life-threatening
☐ hospitalization – initial or prolonged
☐ disability
☐ congenital anomaly
☐ required intervention to prevent permanent impairment/damage
☐ other: ____________

3. **Date of event** (mo/day/yr)

4. **Date of this report** (mo/day/yr)

5. **Describe event or problem**

6. **Relevant tests/laboratory data, including dates**

7. **Other relevant history, including preexisting medical conditions** (e.g., allergies, race, pregnancy, smoking and alcohol use, hepatic/renal dysfunction, etc.)

C. Suspect medication(s)

1. **Name** (give labeled strength & mfr/labeler, if known)
#1 ____________
#2

2. **Dose, frequency & route used**
#1 ____________
#2

3. **Therapy dates** (if unknown, give duration)
from/to (or best estimate)
#1 ____________
#2

4. **Diagnosis for use** (indication)
#1 ____________
#2

5. **Event abated after use stopped or dose reduced**
#1 ☐ yes ☐ no ☐ doesn't apply
#2 ☐ yes ☐ no ☐ doesn't apply

6. **Lot #** (if known)
#1 ____________
#2

7. **Exp. date** (if known)
#1 ____________
#2

8. **Event reappeared after reintroduction**
#1 ☐ yes ☐ no ☐ doesn't apply
#2 ☐ yes ☐ no ☐ doesn't apply

9. **NDC #** (for product problems only)
– –

10. **Concomitant medical products** and therapy dates (exclude treatment of event)

D. Suspect medical device

1. **Brand name**

2. **Type of device**

3. **Manufacturer name & address**

4. **Operator of device**
☐ health professional
☐ lay user/patient
☐ other: ____________

5. **Expiration date** (mo/day/yr)

6.
model # ____________
catalog # ____________
serial # ____________
lot # ____________
other #

7. **If implanted, give date** (mo/day/yr)

8. **If explanted, give date** (mo/day/yr)

9. **Device available for evaluation?** (Do not send to FDA)
☐ yes ☐ no ☐ returned to manufacturer on ____________ (mo/day/yr)

10. **Concomitant medical products** and therapy dates (exclude treatment of event)

E. Reporter (see confidentiality section on back)

1. **Name & address**
phone #

2. **Health professional?**
☐ yes ☐ no

3. **Occupation**

4. **Also reported to**
☐ manufacturer
☐ user facility
☐ distributor

5. If you do NOT want your identity disclosed to the manufacturer, place an " X " in this box. ☐

FDA

Mail to: MEDWATCH
5600 Fishers Lane
Rockville, MD 20852-9787

or **FAX to:**
1-800-FDA-0178

FDA Form 3500 1/96)

Submission of a report does not constitute an admission that medical personnel or the product caused or contributed to the event.

ADVICE ABOUT VOLUNTARY REPORTING

Report experiences with:
- medications (drugs or biologics)
- medical devices (including in-vitro diagnostics)
- special nutritional products (dietary supplements, medical foods, infant formulas)
- other products regulated by FDA

Report SERIOUS adverse events. An event is serious when the patient outcome is:
- death
- life-threatening (real risk of dying)
- hospitalization (initial or prolonged)
- disability (significant, persistent or permanent)
- congenital anomaly
- required intervention to prevent permanent impairment or damage

Report even if:
- you're not certain the product caused the event
- you don't have all the details

Report product problems – quality, performance or safety concerns such as:
- suspected contamination
- questionable stability
- defective components
- poor packaging or labeling
- therapeutic failures

How to report:
- just fill in the sections that apply to your report
- use section C for all products except medical devices
- attach additional blank pages if needed
- use a separate form for each patient
- report either to FDA or the manufacturer (or both)

Important numbers:

• 1-800-FDA-0178	to FAX report
• 1-800-FDA-7737	to report by modem
• 1-800-FDA-1088	to report by phone or for more information
• 1-800-822-7967	for a VAERS form for vaccines

If your report involves a serious adverse event with a device and it occurred in a facility outside a doctor's office, that facility may be legally required to report to FDA and/or the manufacturer. Please notify the person in that facility who would handle such reporting.

Confidentiality: The patient's identity is held in strict confidence by FDA and protected to the fullest extent of the law. The reporter's identity, including the identity of a self-reporter, may be shared with the manufacturer unless requested otherwise. However, FDA will not disclose the reporter's identity in response to a request from the public, pursuant to the Freedom of Information Act.

The public reporting burden for this collection of information has been estimated to average 30 minutes per response, including the time for reviewing instructions, searching existing data sources, gathering and maintaining the data needed, and completing and reviewing the collection of information. Send comments regarding this burden estimate or any other aspect of this collection of information, including suggestions for reducing this burden to:

DHHS Reports Clearance Office
Paperwork Reduction Project (0910-0291)
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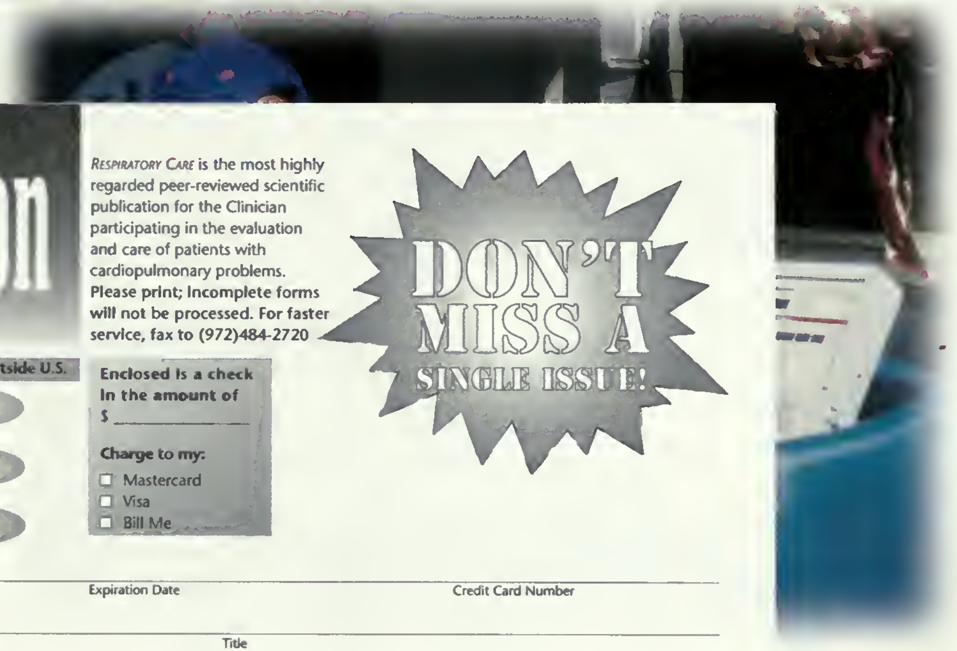

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C ☐ Subacute Care
D ☐ Home Care

III SPECIALTY
1 ☐ Clinician
2 ☐ Perinatal/ Pediatrics
3 ☐ Critical Care
4 ☐ Research
5 ☐ Subacute Care
6 ☐ Diagnostics/ Pulmonary Function
7 ☐ Management
8 ☐ Home Care
9 ☐ Rehabilitation
10 ☐ Education

IV POSITION
A ☐ Department Head
B ☐ Chief Therapist
C ☐ Supervisor
D ☐ Staff Therapist/ Technician
E ☐ Medical Director
F ☐ Educator/Instructor
G ☐ Sales
H ☐ Other (please specify) ________

V ARE YOU AN AARC MEMBER?
1 ☐ Yes 2 ☐ No

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